Psychological mechanisms contributing to alcohol-induced increases in energy intake.

Thesis submitted in accordance with the requirements of the University of Liverpool

for the degree of Doctor of Philosophy by

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April 2021

UNIVERSITY OF LIVERPOOL

INSTITUTE OF POPULATION HEALTH

PSYCHOLOGY (SCIENCE)

DECLARATION IN HIGHER DEGREE THESES

DECLARATION

This thesis is the result of my own work. The material contained in the thesis has not been presented, nor is currently being presented, either wholly or in part for any other degree or qualification.

Abstract

Consumption of alcohol is a significant risk factor for undesirable weight. Previous investigations have demonstrated that acute alcohol consumption reliably increases caloric intake relative to consumption of an alcohol-free comparator, however a complete understanding of the mechanisms contributing to this effect is lacking. Therefore, this thesis investigated the psychological mechanisms underpinning alcohol consumption's effects on eating behaviour. Specifically, it investigated the role of alcohol-induced changes to cognitive control of eating as well as food reward. The thesis also explored whether alcohol-induced changes to food intake and BMI can be explained by a dual-process account of appetite control - an interaction of top-down and bottom-up processes.

Chapter 3 (Studies 1 and 2) investigated whether acute alcohol consumption can impair recall of memories related to a recently consumed meal and whether this affects subsequent food intake. Findings revealed that acute alcohol consumption prior to a lunch meal impaired meal memory recall relative to an alcohol-free drink in Study 2 but not in Study 1. Both studies failed to provide evidence that meal memory recall affects subsequent food intake. Chapter 4 (Studies 3 and 4) investigated whether acute alcohol consumption can enhance food reward, relative to a placebo-alcohol. Study 3 found that an alcohol dose of 0.3 g/kg (grams of alcohol per kilogram of bodyweight) did not enhance food reward (measured using self-report scales and an attentional bias task) or increase food intake. However, Study 4 showed that a dose of 0.6 g/kg, acute alcohol consumption enhanced food reward and food intake relative to the placebo. Contrary to predictions of the dual-process account, the interaction between trait motor impulsivity and change in food-related attentional bias between drink conditions did not significantly predict change in food intake. Finally, Chapter 5 (Study 5) examined whether change in BMI over a 12-month period is predicted by change in drinking behaviour, and whether trait motor impulsivity moderates this effect. Findings did not support these predictions.

Overall, the findings of this thesis demonstrate that acute alcohol consumption does affect important cognitive factors implicated in appetite control, however there is no evidence to suggest that this contributes to alcohol-induced increase in food intake. Results also indicate that alcohol-induced enhanced food reward and increases in food intake may be dose-dependent, whereby lower doses of

alcohol may be insufficient at increasing food intake. The dual-process model of eating behaviour did not appear to explain alcohol-induced change to food intake or BMI in these studies. Further research regarding the importance of cognitive and reward-based mechanisms within the context of alcohol-induced food intake and BMI is now warranted.

Acknowledgements

Thank you to my supervisory team, Dr Charlotte Hardman, Dr Paul Christiansen and Dr Abi Rose for all of your help and support over the past four years. I have gained a wealth of knowledge and experience which I will carry with me throughout my career. A particular thanks to my primary supervisor Dr Charlotte Hardman whose brilliance in research has greatly contributed towards the completion of this thesis. Thank you also to Professor Peter Rogers for providing me with an introduction to the research area, inspiring me to pursue this research further, and for introducing me to Dr Charlotte Hardman.

I have also had the privilege of working with brilliant people within the department. Thank you Dr Eric Robinson for providing me the opportunity to work on additional projects during my PhD. Thank you also to the demonstrators who have been a great source of support and help, particularly my office mates, Anna, Rachel and Sarah. Anna, thank you for giving me such a welcoming and friendly introduction to the department.

I would also like to thank everyone who participated in my studies. This research would not have been possible without their willingness to consume a substantial amount of vodka during working hours, all in the pursuit of science – a truly selfless act.

Lastly, I would like to thank my family for all of their support throughout my PhD and also encouraging me to attend University – Mum, Dad, Hazel, Jamie and many others, thank you.

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Chapter 1: General Introduction

Chapter outline

In this chapter, theories of appetite control will be outlined, focusing on contemporary accounts of eating behaviour which state that appetite control is determined by an interplay between metabolic, reward and cognitive systems. The chapter will then provide an overview of theories and supporting literature specifically relating to reward and cognitive processes, and the integration of these processes, in determining of eating behaviour. Next, literature on how acute alcohol consumption can affect food intake will be discussed, focusing on both previously tested (but inconclusive) mechanisms as well as novel ways in which alcohol intoxication (defined in this thesis as a blood alcohol level > 0) may affect food intake. Lastly, literature regarding the longer-term associations between alcohol consumption and measures of adiposity will be reviewed, including the identification of limitations within existing literature which may be contributing to inconsistent findings.

1.1. The prevalence of obesity

Increases in body mass index (BMI) and rates of obesity (defined as a BMI ≥ 30) are commonplace throughout the world. Globally, in 1995 an estimated 200 million adults had obesity, this figure rose to 300 million by 2000 (House of Commons Health Committee, 2004) and is projected to rise to 573 million by 2030 (Kelly et al., 2008). In the US, between 2011 and 2014, the prevalence of obesity was estimated to be 36.5% (Ogden et al., 2015). In the UK, data from the Health Survey for England 2017 estimates that the prevalence of adults with overweight and obesity are 28.7% and 35.6% respectively (House of Commons, 2019).

Obesity is believed to cause an estimated 9,000 premature deaths each year in the UK (Kelly et al., 2009) and is associated with a number of health problems and diseases including heart disease, stroke, cancer and diabetes (Agha & Agha, 2017). Additionally, obesity is associated with a reduction in quality of life which includes an increased risk of back pain, shortness of breath and reduced

mobility (World Health Organisation, 2007). Furthermore, individuals with obesity face stigma which has been shown to be associated with depression and low self-esteem (Carr & Friedman, 2005; Farrow & Tarrant, 2009).

There is also a significant economic cost of obesity on society and health services. It is estimated that in the UK, £6.1 billion was spent on overweight and obesity-related health issues between 2014 and 2015 as well as an estimated cost of £27 billion to wider society (Public Health England, 2017). Projections also estimate that by 2050, UK-wide NHS costs which are attributed to overweight and obesity will reach £9.7 billion, with wider societal estimates reaching £49.9 billion by the same year (Public Health England, 2017).

Obesity results from an energy surplus (i.e., over-consumption of energy). The aetiology of obesity is complex, resulting from a combination of both internal and external factors. Importantly, a key determinant of overeating comes from individual differences in appetite control.

1.2. An integrative model of appetite control: combining metabolic, reward and cognitive processes.

Appetite control is a highly complex construct which is determined by multiple systems. Traditional theories of appetite control argue that processes contributing to appetite control are often separate and treated in parallel (e.g., the dichotomy between homeostatic and hedonic systems) (Waterson & Horvath, 2015). However, more contemporary accounts now acknowledge that eating behaviour is determined through an interaction of systems. Higgs et al. (2017) propose a model which states that metabolic, reward and cognitive processes interact with each other to determine appetite control. Crucially, this account of eating behaviour also argues that central traditional processes (metabolic and hedonic) interact with each other as well as integrate with higher-level processes, including cognitive factors such as attention and memory (Higgs, 2016).

1.2.1. Metabolic control of eating behaviour

The ability of organisms to successfully maintain homeostatic metabolic control of appetite is essential for survival. Metabolic control is achieved through constant communication between metabolic states and activation of brain regions, which produce an appropriate motor response.

Several brain regions have been implicated in the neural maintenance of energy balance, particularly the hypothalamus. Research dating back 70 years found that lesions to the ventromedial hypothalamus increases feeding in rats, whereas lesions to the ventrolateral hypothalamus produces reduced feeding (Anand & Brobeck, 1951a; Anand & Brobeck, 1951b). Within the hypothalamus, neurons expressing neuropeptide Y (NPY) and agouti-related protein (AgRP) have been shown to increase eating and reduce weight loss (Gropp et al., 2005; Luquet et al., 2005), whereas cells expressing Pro-opiomelanocortin (POMC) and cocaine-and-amphetamine-regulated transcript (CART) produce the opposite effect (Gropp et al., 2005; Xu et al., 2005).

Peripheral signals which influence food intake and energy expenditure broadly fall under two categories: satiation signals and adiposity signals. Satiation signals produce feelings of fullness and satiety and are secreted from cells in the wall of the Gastrointestinal tract when food is being digested or absorbed. Numerous hormones have been shown to affect satiation as part of this response, these include Cholecystokinin (CCK) and glucagon-like peptide 1 (GLP-1) - release of these hormones is associated with a reduction in food intake (Antin et al., 1975; Turton et al., 1996). Conversely, other hormones have the opposite effect on satiation when secreted. For example, when released, ghrelin activates both AgRP and NPY cells (Betley et al., 2015; Chen et al., 2015), inhibits POMC cells (Cowley et al., 2003) and increases food intake (Nakazato et al., 2001).

Unlike the phasic secretion of satiation signals, adiposity signals are tonically active and are secreted in direct proportion to body fat. These hormones include insulin, which is secreted from pancreatic cells, and leptin, which is secreted from adipose tissue. Both of these hormones have direct action on cells within the hypothalamus, including the inhibition of activity to NPY and AgRP cells and the stimulation of activity to POMC which can then reduce food intake (Cowley et al., 2001).

1.2.2. Hedonic systems of appetite control and food reward

Hedonic systems of appetite control and eating behaviour focus on reward-driven behaviours which can be initiated by external influences in the absence of a physiological need to consume food (i.e., a caloric deficit). For example, environmental factors such as exposure to palatable food cues can increase the desire to consume the same cued foods (Fedoroff et al., 2003; Ferriday & Brunstrom,

2008; Ferriday & Brunstrom, 2011). Also, factors relating to the food itself, such as palatability, have shown to increase food intake in the absence of change in nutritional value (De Graaf et al., 1999; Yeomans, 1996; Yeomans & Symes, 1999). This suggests that factors independent of metabolic signals can alter eating behaviour and stimulate food intake.

This type of eating behaviour is directly related to the concept of food reward - defined as the momentary value of a food (Rogers & Hardman, 2015). Food reward is formed of multiple behavioural constructs which derive from both hedonic and homeostatic systems. Rogers and Hardman (2015) argue that food reward consists, in part, of liking of a food – defined as the pleasantness of the taste and flavour of a food – and physiological hunger. Support for this account of food reward comes from evidence demonstrating that these two components independently predict self-reported 'desire to eat' ratings – a measure used to capture food reward in humans (Rogers & Hardman, 2015).

Others have proposed that food reward consists of two neurologically separate components – 'liking' and 'wanting' – the latter referring to the incentive value of a food (Berridge 1996; 2007). The Incentive Sensitization Theory (IST) (Robinson & Berridge, 1993), originally a neurocognitive model of addiction, proposes that the incentive value of a drug is gained through a process of learning. The theory has subsequently been applied to eating behaviour (Appelhans et al., 2016), arguing that consumption of foods elicits dopamine transmission in reward-related brain regions, the magnitude of this dopamine transmission increases each time the behaviour is repeated – the response becomes sensitized. After multiple occurrences of this sensitized release of dopamine into reward-related regions, an increase in motivational appeal towards food is produced which is expressed through an increase in cravings or a desire to consume foods. The theory also posits that cues related to foods gain motivational properties and become attention-grabbing and 'wanted'.

A key component of the IST is that a stimulus can be 'wanted' in the absence of it being 'liked', and therefore, these components of food reward are argued to be neurologically dissociable. Through use of animal models, a large body of research has attempted to map out brain regions implicated in 'wanting' and 'liking'. The component of 'liking' and pleasure derived from

consumption of foods is localised to circuits within the nucleus accumbens, ventral pallidum and brainstem parabrachial nucleus, which become activated by opioid release (Kringelbach, 2005; Peciña et al., 2006; Smith & Berridge, 2007; Smith & Berridge, 2005; Smith et al., 2009). There is a degree of overlap with regions implicated in 'wanting', as increased opioid transmission within some areas of these hotpots also increase 'wanting' (Smith et al., 2009). $GABA_A$ receptors within the ventral pallidum, however, appear to target 'wanting' without stimulating 'liking' as these have been shown to activate 'wanting' and increase food intake (Stratford et al., 1999), without altering or increasing hedonic reactions to taste (Shimura et al., 2006). The activation of 'wanting' is also linked with dopaminergic activity within the mesolimbic pathway, which projects from the ventral tegmental area to the nucleus accumbens (Berridge, 2007). Taken together, evidence from animal models show a degree of neurological distinction between 'wanting' and 'liking'.

Importantly, in humans, the feasibility of separately capturing and measuring 'wanting' in isolation of other components of food reward, such as 'liking' and hunger, has been questioned (Havermans, 2011, 2012; Pool et al., 2016). Without being able to undertake the same neurological investigations as in animal models, a great deal of human research has implemented behavioural measures to capture different components of food reward. In line with predictions of the IST, attempts have been made to create a measure which can demonstrate dissociative behaviour between 'liking' and 'wanting' in humans. For example, researchers have implemented preference tasks (Finlayson et al., 2007, 2008) and memory measures (Lemmens et al., 2009) to capture 'wanting' in isolation. However, concerns have been raised regarding the use of these measures, highlighting the inherent difficulty of capturing 'wanting' without such a measure also being influenced by current levels of explicit liking (Havermans, 2011; Rogers & Hardman, 2015). As an example, Rogers and Hardman (2015) reported that desire-to-consume ratings were significantly independently predicted by both physiological hunger and liking, suggesting that behavioural measures of 'wanting' cannot be easily separated from 'liking'. Therefore, within humans, behavioural measures which aim to capture 'wanting' should best be thought of as capturing overall levels of food reward instead.

1.2.3. Metabolic control and food reward

Food reward and hedonic systems of appetite control appear not to be separate from homeostatic systems, but interact with each other. As previously mentioned, energy deprivation increases the momentary value of food, which is expressed through increased responding towards such cues (i.e., attentional bias) and an increase in activation of reward-related brain regions (Führer et al., 2008; Haase et al., 2009), whereas the sensation of fullness produces the opposite effect in reward-related regions (Thomas et al., 2015). Specific metabolic hormones have also been implicated in the modulation of food reward. For example, administration of leptin reduces activity in the mesolimbic dopamine system (Fulton et al., 2006). Conversely, microinjections of ghrelin to the VTA produces food-motivated behaviour in animals (Skibicka et al., 2011), whereas activation of GLP-1 receptors within the VTA and nucleus accumbens core produces reduced intake of palatable foods (Alhadeff et al., 2012). Taken together, extensive research has demonstrated that activity related to metabolic control of appetite also affect food reward processing.

1.2.4. The role of cognitive factors in appetite control

A growing body of research suggests that appetite control and motivation to consume food can depend on multiple cognitive factors such as learning, memory and attention (Higgs, 2016), which are thought to affect food-related decision making (Rangel & Hare, 2010). For example, when presented with a food, expectations of the taste and how satiating the food will be, come from previously learned experiences and learned associations of eating (Dickinson, 2012).

Cognitive control of eating behaviour also allows food choice to be goal-directed and adaptable. Predicted outcomes of a behaviour are computed in the ventromedial-prefrontal cortex (vmPFC) which then interacts with the dorsolateral prefrontal cortex (dlPFC) in order to select an action (Hare et al., 2009). The interaction of these brain regions when making food choices allows for eating behaviour to be goal-directed and not solely cue-driven. The ability to produce goal-directed eating behaviour is affected by multiple factors, such as whether long-term goals can be retrieved from memory and then placed in one's attention (Hare et al., 2010; Whitelock et al., 2018b). Other factors include the degree to which cues relating to competing goals are present in the environment,

whether we have sufficient available cognitive resources to focus on these goals, and whether or not we are distracted by environmental stimuli which may interfere with these cognitive demands, such as viewing television (Braude & Stevenson, 2014; Ward & Mann, 2000).

1.2.5. A dual-process account of eating behaviour

Theories of appetite control also argue that cognitive control and reward systems interactively predict BMI and food intake. For example, dual-process models of eating behaviour argue that food intake and weight is largely determined by an interaction of top-down and bottom-up processes (Appelhans, 2009). Strack and Deutsch (2004) state that decision making processes operate through two separate but parallel systems: the reflective system (a top-down process) and the impulsive system (a bottom-up process). The reflective system relates to cognitive processes and operates through knowledge about values and goals, and has limited capacity, meaning that this system can be disrupted under circumstances where cognitive capacity is reduced. The reflective system can be captured using measures of impulsivity (i.e., trait impulsivity using self-report questionnaires, inhibitory control, and impulsive decision making). The impulsive system involves reward processing and implicitly processes information outside of conscious awareness and elicits behaviour based on motivational orientation. This system is measured using tasks which capture appetitive responding to food such as food reward responsivity. In the context of eating behaviour, dual-process models argue that the tendency to overeat is determined by an interaction of both the reflective and impulsive system.

Several studies have demonstrated how these two components interactively predict eating behaviour and weight change. For example, Price et al. (2015) showed that the degree to which food reward responsivity predicts BMI is moderated by levels of inhibitory control (a measure of top-down processing) – food reward responsivity predicted BMI only when impulsiveness was high (indicative of a weaker reflective system). Both Jansen et al. (2009) and Nederkoorn et al. (2009) have shown poorer response inhibition to be related to overeating and BMI, only when desire to eat is high. Nederkoorn et al. (2010) showed that weight gain over a 1-year period is predicted by food reward sensitivity, but only for individuals who performed poorly at a response inhibition task. Similarly,

Rollins et al. (2010) found food reward sensitivity to be predictive of palatable food intake only in individuals who showed diminished inhibitory control. Appelhans et al. (2011) revealed that food reward sensitivity and inhibitory control interact to predict food intake in individuals with obesity. Kakoschke et al. (2015) found a significant interaction between an approach bias towards food and inhibitory control on *ad libitum* unhealthy snack food intake.

Collectively, these findings suggest that an interaction of top-down and bottom-up processes significantly predict eating behaviour and adiposity. In the next two sections, central measures implicating bottom-up and top-down processes within the context of eating behaviour will be reviewed – namely attentional bias and impulsivity, respectively.

1.2.6. Attentional Bias

Attentional bias is defined as the tendency for a specific cue to capture attention (Field et al., 2016). In the context of eating behaviour, food-related attentional bias can capture top-down processes. For example, evidence suggests that food-related attentional biases can increase when holding food cues in working memory (Higgs et al., 2015; Higgs et al., 2012).

As well as top-down processes, food-related attentional biases are also implicated in bottom-up processing and are suggested to be a proximal measure of food reward (Field et al., 2016). Attentional bias towards cues are likely adaptive and would help to forage for food in environments of food scarcity (Nijs et al., 2010). However, now that many humans live in 'obesogenic' environments, characterised with an abundance of energy dense food and ubiquitous food cues, a heightened responsivity towards these cues may produce risk of overeating in certain individuals (Nijs & Franken, 2012). Food-related attentional biases can be measured using indirect attentional behaviours (e.g., response latency towards certain cues), as assessed with tasks such as the modified Stroop task visual probe task, or by using more direct measures of attention placed towards certain cues during completion of a task, such as capturing visual attention using eye-tracking methodology.

Many theoretical models aiming to explain the cause of attentional bias exist. For example, based on the previously mentioned Incentive Sensitization Theory, cues relating to food gain

incentive salience through repeated exposure to the rewarding effects of food consumption and become sought after and are more attention-grabbing in one's environment.

Although eating is a universal behaviour which everyone experiences, individuals with obesity have been argued to display stronger attentional biases towards palatable food cues compared with healthy weight individuals, possibly due to the presence of lifestyle interventions (e.g., diet adherence) within individuals with obesity which then cause food cues to act as a "motivational magnet" (Appelhans et al., 2016).

Several studies have investigated whether the magnitude of food-related attentional biases differ between individuals with obesity and healthy weight. Research to date has yielded an inconsistent pattern of findings. For example, Werthmann et al. (2015) reported 11 studies which had compared food-related attentional bias between individuals with overweight/obesity and healthy-weight individuals, however findings were mixed. Furthermore, evidence from two meta-analyses suggest that attentional bias towards food cues does not differ across weight (Hagan et al., 2020; Hardman et al., 2020). Hagan et al. (2020) showed that healthy-weight individuals did not differ from individuals with overweight and obesity on both automatic and maintained attention using a visual-probe task, attentional bias using the modified Stroop task, and attentional bias using gaze-direction and gaze-duration biases. Similarly, Hardman et al. (2020) showed that BMI was unrelated to food-related attentional biases, and also found no effect of weight status on attentional biases.

Other models of attentional bias argue that the amount of attention placed towards food cues is indicative of appetitive motivational states. Links between attentional bias and subjective cravings have been found in other types of attentional bias, with one meta-analysis showing a small but significant relationship between subjective craving of a substance and attentional bias (r = .19; Field et al., 2009). Regarding food-related attentional bias, numerous studies have shown a link between AB and subjective hunger and food craving (Castellanos et al., 2009; Gearhardt et al., 2012; Graham et al., 2011; Mogg et al., 1998; Nijs et al., 2010; Schmitz et al., 2014; Tapper et al., 2010; Werthmann et al., 2013; Werthmann et al., 2011). Furthermore, the meta-analysis by Hardman et al. (2020) revealed that food-related AB was significantly (but weakly) related to subjective cravings (r = .13),

hunger ratings (r = .05) and food intake (r = .09). Collectively, these findings suggest that food-related attentional biases better reflect within-subject fluctuations of motivational states rather than between-subject differences (Field et al., 2016).

1.2.7. Impulsivity, food intake and BMI

Impulsivity can be defined as having a predisposition towards the initiation of rapid and unplanned reactions to stimuli which may produce negative consequences to the individual (Moeller et al., 2001). It is a multifaceted construct, consisting of several dimensions when assessing its factor structure (Christiansen et al., 2012; Reynolds et al., 2006a). Impulsivity can be measured using self-report and behavioural measures. Behavioural measures of impulsivity capture state-like behaviours which can fluctuate over time. Examples include the delay discounting task which measures delayed gratification, and inhibitory control which relates to one's ability to inhibit a behavioural impulse (Houben et al., 2012). Conversely, self-report measures are thought to measure stable, trait impulsivity. Such measures include the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001) which consists of the following subscales: urgency (tendency to behave rashly when experiencing negative emotions), lack of premeditation (acting without thinking), lack of perseverance (inability to stay focused on a task), and sensation seeking (tendency to seek thrilling experiences). A second measure is the Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995), subscales of this consists of: non-planning (lack of future thinking), motor (acting without thinking), and attentional impulsivity (inability to focus attention).

Overall, evidence suggests that impulsivity is inconsistently linked with overeating and BMI across different types of measures. For subscales of the UPPS, Murphy et al. (2014) showed that only premeditation was significantly correlated with BMI. Conversely, Ellickson-Larew et al. (2013) found that urgency, but no other UPPS subscales, were significantly associated with BMI. For the BIS-11, motor impulsivity has been shown to be higher in groups with binge-eating behaviours compared with controls (Nasser et al., 2004; Rosval et al., 2006). Lyke and Spinella (2004) showed that the attentional and motor impulsivity subscales were positively correlated with the disinhibited eating subscale of the Three-Factor Eating Questionnaire (Stunkard & Messick, 1985). An additional two

studies have shown that the subscale of motor impulsivity, but no other BIS-11 subscales or total score, were positively associated with BMI (Price et al., 2015; Van Koningsbruggen et al., 2013).

Regarding behavioural measures, many studies have demonstrated that heavier delay discounting (i.e., a preference towards small, immediate rewards over long-term larger rewards) is related to overeating and BMI, however this effect appears to be small and is often inconsistent across studies, possibly due to insufficient sample sizes (Appelhans et al., 2011; Bickel et al., 2014; Davis et al., 2010; Epstein et al., 2014; Jarmolowicz et al., 2014; Nederkoorn et al. 2006; Weller et al., 2008). Regarding response inhibition, studies have shown that inhibitory control can predict BMI and overeating. For example, Price et al. (2016) showed that performance on the Go/No-Go task (a measure of inhibitory control) predicted snack intake. Batterink, Yokum and Stice (2010) found that BMI was positively correlated with impulsivity (also measured using a Go/No-Go task) and inversely correlated with neural activation of brain regions implicated in response inhibition. Response inhibition has been shown to differ across weight statuses, for example performance on the stop signal task is impaired in individuals with obesity compared with lean individuals (Nederkoorn et al., 2006). However, similar to delay discounting, this effect appears to be inconsistent, as other studies have been unable to show that response inhibition predicts overeating and BMI (Houben et al., 2014; Nederkoorn et al., 2009).

Collectively, findings show that impulsivity is inconsistently related to eating behaviour and BMI, suggesting that impulsivity alone is a poor predictor of these outcome measures. Instead, impulsivity may best be considered as a predictor of eating behaviour within the context of a dual-process account of appetite control, which argues that an interaction of top-down and bottom-up processes predict eating behaviour, as evidenced by previous research (Jansen et al., 2009; Nederkoorn et al., 2009), reviewed in Section 1.2.5.

1.2.8. Episodic memories of food and food reward

Another important cognitive factor shown to influence components of food reward and eating behaviour is that of memory relating to food. Enjoyment of a food can increase when memories of an enjoyable previous experience of a meal are primed. For example, Robinson et al. (2011) demonstrated that when participants were asked to recall a previous occasion when vegetables were consumed and enjoyed, ratings of expected enjoyment of vegetables increased. In another investigation, Robinson et al. (2012) showed that when participants were asked to write down the enjoyable aspects of a meal which they had just consumed, the remembered enjoyment of the meal increased. Furthermore, in a second study by Robinson et al. (2012) this same manipulation resulted in greater food choice and intake of a meal in which the positive aspects had been rehearsed.

In addition to priming and altering the remembered aspects of a meal, studies have investigated how boosting or reducing the quality of a meal-related episodic memory can affect satiety and food intake. A large body of research has demonstrated that impairments to episodic memories relating to recently consumed food can alter subsequent food intake. For example, animal research has demonstrated that lesions to the hippocampal region results in hyperphagia and weight gain (Clifton et al., 1998; Davidson et al., 2005). Further, evidence from amnesic patients has demonstrated that individuals who have an impaired ability of reporting memories for recent eating also display evidence of overeating (Higgs et al., 2008b).

Within neurologically intact humans, enhancing memories of a recently consumed meal has been shown to reduce subsequent food intake (Higgs, 2002). Using a recall paradigm, participants were given a fixed lunch to consume and then after a 2-3-hour delay, participants returned to the laboratory and were asked to recall what they remembered about the lunch meal. In a separate condition, participants were asked to recall details of a lunch meal consumed the previous day. Findings revealed that subsequent food intake after the 2-3-hour break was reduced when participants recalled details of the recent lunch, but not when participants recalled details of lunch on the previous day, suggesting that only cueing of recent eating episodes affects subsequent food intake.

Similarly, many studies have investigated whether increasing the level of attention placed on food can facilitate the encoding process, thereby resulting in higher quality memories of the eating episode, and whether this can reduce later food intake. Evidence for this suggestion is mixed. Many studies have demonstrated that focusing on food while eating is associated with lower levels of subsequent intake (Higgs, 2015; Higgs & Donohoe, 2011; Robinson et al., 2014b; Seguias & Tapper, 2018). However, other studies have failed to demonstrate that focused attention produces a reduction in food intake (Tapper & Seguias, 2020; Whitelock et al., 2019; Whitelock et al., 2018a). Importantly however, recent investigations have raised questions regarding whether increased focused attention facilitates the encoding of episodic memories, as evidence has failed to show a difference in meal-memory recall between participants who did and did not use focused attention during consumption of a lunch meal (Seguias & Tapper, 2018).

Another approach to examine the role of episodic memory is by disrupting the encoding process of memory formation. This has been tested by distracting participants from eating by including a secondary activity for participants to engage with whilst eating. It is argued that because attention is divided by the distracting activity and the meal, encoding of memories relating to the meal will be poorer, which will then increase food intake due to a reduction in the inhibitory effects of remembering a meal. Indeed, participants who consume a lunch whilst watching television have been shown to consume more afternoon snacks, compared with when participants consume lunch with no distraction (Higgs & Woodward, 2009). This effect has been replicated by subsequent research which has used the same as well as different forms of distraction (i.e., playing a computer game) (Higgs, 2015; Mittal et al., 2011; Oldham-Cooper et al., 2011). Importantly, in these studies, meal memory was poorer within the distraction conditions, suggesting that the quality of the meal memory may have contributed to the amount of food eaten.

Brunstrom et al. (2012) investigated how episodic memory of a meal can affect hunger and fullness ratings over the course of three hours. In this study, participants were split into two conditions and were shown either a 300 ml or 500 ml portion of soup, which they were told they would consume. Half of the participants in each condition then consumed either 300 ml or 500 ml of soup (creating a

total of four conditions crossed by expected and actual consumption). Findings revealed that hunger ratings immediately after consumption of the lunch were influenced by actual soup consumption – those who consumed the larger portion of soup had lower hunger ratings. However, hunger ratings two and three hours afterwards was predicted by the perceived amount consumed. This suggests that after a delay, hunger was directly affected by episodic memories of a meal consumed.

1.2.9. Summary

Collectively, the reviewed research has outlined the role of food reward and cognitive processes in appetite control, showing that these both independently and interactively predict food intake and changes to adiposity. Importantly, lifestyle behaviours which can influence these processes may therefore be risk factors for increased food intake and weight gain, one such factor is alcohol consumption. In the next section, research which has investigated the role of acute alcohol consumption in altering energy intake will be reviewed. Specifically, the next section will review whether acute alcohol consumption can increase energy intake and review the potential mechanisms which may explain any observed increase in caloric intake, with a focus on reward and cognitive processes.

1.3. Alcohol Prevalence in the United Kingdom

Alcohol is considered the third biggest global risk for burden of disease by the World Health Organization (World Health Organization, 2009) and is implicated as a casual factor in several medical conditions, including cancers, liver cirrhosis, cardiovascular diseases and gastrointestinal diseases. (Department of Health, 2016).

In 2017/2018, data from the NHS estimated that there were 358,000 cases where the main reason for hospital admission was attributable to alcohol and 1.2 million hospital admissions where the reason for admission was either a primary or secondary diagnosis linked to alcohol, representing 7.4% of all hospital admissions (NHS, 2018). Of those admitted, around two-thirds were male, and 51% of admissions were related to cardiovascular disease. Furthermore, In England in 2018, there were 5,698 alcohol-specific deaths, an increase of 7% since 2008 (NHS, 2018). Alcohol-specific deaths were greatest in the 50-59 age bracket, with 77% of deaths belonging in the age range 40-69

and 67% of alcohol-specific deaths were for men. Of these alcohol-specific deaths, alcoholic liver disease accounted for 79%, whereas 10% were related to mental and behavior disorders resulting from alcohol use. Alcohol-specific death rates also vary by socio-economic position, with such deaths shown to be highest in areas with the greatest deprivation and lowest in areas with the lowest levels of deprivation (NHS, 2018).

1.4. Alcohol metabolism and sex differences

Most commonly, alcohol is metabolised through pathways which involve two enzymes – alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH). ADH breaks alcohol down into acetaldehyde, which is then broken down into acetate, then acetate is broken down into water and carbon dioxide for elimination (Edenberg, 2007). Alcohol metabolism primarily occurs in the liver, but also occurs to a lesser extent in the pancreas, brain and gastrointestinal tract (Zakhari, 2006). Several factors can affect variation of alcohol metabolism. One such factor is genetic variation of the ADH and ALDH enzymes. For example, ADH variants ADH1B*2 and ADH1B*3 metabolise alcohol to acetaldehyde efficiently, resulting in greater levels of acetaldehyde (Crabb, 1995).

Alcohol metabolism has also been shown to differ between males and females. Findings suggest that women eliminate significantly more alcohol per unit of lean body mass per hour, compared to men (Ammon et al., 1996; Thomasson et al., 1995). However, women have lower proportion of body water (Ritz et al., 2008) and a greater proportion of body fat compared with men (Gallagher et al., 1996; Jackson et al., 2002; Womersley & Durnin, 1977). Both of these factors contribute to a greater blood alcohol concentration in women than men after consuming equivalent amounts of alcohol (Mumenthaler et al., 1999), as body water helps to disperse alcohol, whereas body fat helps to retain alcohol. Studies have suggested that sex differences in peak concentrations following consumption of low alcohol doses is due to first-pass metabolism in the gastrointestinal tract (Baraona et al., 1998; Frezza et al., 1990), however other studies have failed to show this sex difference (Ammon et al., 1996).

Sex differences in hormonal levels may also affect alcohol absorption and metabolism. For example, increased levels of progesterone (a steroid hormone involved in the menstrual cycle) is

associated with faster alcohol elimination rates in women but not men (Dettling et al., 2008). However, other research has found that menstrual cycle-related changes in estradiol and progesterone not to affect alcohol metabolism or account for sex differences in metabolism (Lammers et al., 1995; Mumenthaler et al., 1999). The effect of oral contraceptives use on alcohol pharmacokinetics has also been investigated, with findings showing that contraceptive use is positively associated with acetaldehyde levels (Eriksson et al., 1996; Jeavons & Zeiner, 1984). Studies have also reported that oral contraception use decreases alcohol elimination rate (Jones & Jones, 1984), whereas other research has found no effect of oral contraceptives on alcohol metabolism (King & Hunter, 2005; Sarkola et al., 2002). Therefore, currently research is mixed as to whether hormonal levels have a significant effect on alcohol metabolism.

Due to sex differences in alcohol metabolism, research has also been conducted to investigate whether differences in alcohol-induced changes to cognitive performance exist between men and women. Niaura et al. (1987) investigated sex differences regarding alcohol's effect on short-term memory impairments and revealed that after consumption of an alcoholic drink, women took longer to recover from short-term memory impairments than men. Similarly, acute alcohol consumption has been shown to produce short-term memory impairments to a greater degree in females than in males (Jones & Jones, 2014). However, another study showed that women displayed greater alcohol-induced impairment of delayed recall, but not immediate recall, compared with men (Jones & Jones, 1976). Women have also been shown to respond more slowly on cognitive decision making tasks than men after alcohol consumption (Haut et al., 1989). The extent to which sex differences in alcohol-induced changes to cognitive performance exist, may be dose-dependent. Mills and Bisgrove (1983) found that women performed significantly worse than men on a divided attention task at a blood alcohol content of 0.06%, but there was no gender differences at a blood alcohol content of 0.03%. Taken together, findings demonstrate that alcohol may display greater impairments on certain aspects of cognition in women, compared to men.

1.5. The role of acute alcohol consumption in altering energy intake

The macronutrient alcohol has a high energy density of 7.1 kcal/g, second only to fat (9 kcal/g). Given that energy consumed from liquid preloads is poorly compensated for, relative to semi-solid and solid preloads (Almiron-Roig et al., 2013), consumption of alcohol may be a likely cause of weight gain, if calories from alcoholic beverages are not compensated for. Furthermore, evidence suggests that alcohol may be the least satiating macronutrient (Westerterp-Plantenga & Verwegen, 1999). Alcohol also inhibits fat oxidation (Suter et al., 1992), meaning that frequent levels of alcohol consumption can lead to higher body fat after long-term use.

Several laboratory studies have investigated how alcohol can affect caloric intake, relative to an alcohol-free control with two main focuses: 1) to see whether consumption of an alcoholic drink produces greater total caloric consumption relative to an alcohol-free beverage and 2) to see whether consumption of an alcoholic drink increases subsequent food intake relative to an alcohol-free control. To date, two meta-analyses have been conducted which focus on these findings, both with similar conclusions (Chapman et al., 2012; Kwok et al., 2019). Chapman et al. (2012) conducted a meta-analysis on 14 studies which compared an alcohol preload with an alcohol-free control preload on subsequent food intake, and showed that consumption of an alcoholic drink significantly increases food intake. More recently, Kwok et al. (2019) conducted a similar meta-analysis, but also differentiated between total caloric intake (the combined number of calories consumed from the test drink and subsequent eating episode) and food caloric intake (the number of calories consumed only from a subsequent eating episode). Furthermore, subgroup analyses were performed to examine whether such an effect differed between sex, whether the dose of the alcoholic drink moderated the magnitude of the effect, and to examine whether the use of different control drinks (an energy-containing drink or a control drink with negligible or no energy) influenced the effect. Findings showed that firstly, an effect of acute alcohol consumption on both total and food caloric intake was found – with greater caloric intake after consumption of an alcoholic drink - when using an energy-containing control drink or a control with little-to-no energy content. Secondly, splitting studies by a low dose (< 30 g of alcohol or < 0.6 g/kg) and a high dose (≥ 30 g of alcohol or ≥ 0.6 g/kg) revealed

that total caloric intake increased after consumption of an alcoholic drink regardless of the dose, however food intake only increased after consumption of alcohol at a low dose. The authors suggest that this latter null finding at higher doses may have occurred due to substantial heterogeneity between the reviewed studies, which have used a mixture of fixed and body-weight dependent doses, as well as different alcohol types and variation in the interval length between alcohol consumption and food intake. Lastly, the subgroup analysis of sex revealed that both total caloric and food caloric intake increased after consuming alcohol in both male and female-only studies. Overall, the meta-analysis showed that on average, consumption of an alcoholic beverage relative to an alcohol-free comparator increased food intake by 343 kJ and total caloric intake by 1072 kJ.

1.5.1. Potential mechanisms producing an acute increase in food intake

1.5.1.1. Food Reward

Neurological evidence shows that acute alcohol consumption produces change within reward-related brain regions. It produces pharmacological actions within the brain which are implicated in reward processing behaviours, including changes to the binding of $GABA_A$ receptors (Lobo & Harris, 2008), and changes in the opioid and dopaminergic systems (Berridge, 1996; Yeomans & Gray, 2002). Acute alcohol consumption also increases dopamine release and glucose metabolism in the ventral striatum, including the nucleus accumbens (Boileau et al., 2003; Volkow et al., 2008; Yoder et al., 2007). Additionally, acute alcohol consumption produces hyperactivity of agouti-related protein neurons (Cains et al., 2017) and also influences hormones linked with satiety, including the inhibition of leptin and GLP-1 hormones (Raben et al., 2003; Röjdmark et al., 2001).

Focusing specifically on food reward, human research has focused on alcohol-induced changes to this behaviour using both explicit and implicit measures. Regarding explicit measures, studies have used self-report hunger, liking of a food and desire to eat a food as proxies for changes in food reward *after* consumption of an alcoholic beverage and *before* a subsequent eating episode. To date, findings are mixed as to whether acute alcohol consumption increases food reward relative to an alcohol-free control. For hunger ratings, Caton et al. (2004) showed that hunger levels were elevated after consumption of 32 g of alcohol, relative to 8 g of alcohol, suggesting a dose-dependent response.

However, several studies have failed to show such an effect (Eiler et al., 2015; Hetherington et al., 2001; Poppitt et al., 1996; Rose et al., 2015; Westerp-Plantenga & Verwegen, 1999; Yeomans & Phillips, 2002). Eiler et al. (2015) found no change in hunger ratings between intravenous infusion of alcohol in saline (achieving a breath alcohol concentration of 50 mg%) or saline alone. Hetherington et al. (2001) found no difference in appetite ratings between consumption of an alcohol-free beer and a beer containing 24 g of alcohol. Poppitt et al. (1996) failed to show changes in hunger ratings after a preload containing 30.6 g of alcohol, compared with an alcohol-free preload, a carbohydrate-containing preload, and water preload. Rose et al. (2015) found a nonsignificant difference in appetite ratings between a 0.6 g/kg dose of alcohol and an alcohol-free placebo. Yeomans and Phillips (2002) found no differences in hunger ratings between 15 g of alcohol, a non-alcoholic beer and water. One study (Westerp-Plantenga & Verwegen, 1999) found that hunger ratings *decreased* after consumption of a 37.4 g alcohol preload of wine and a 34 g alcohol preload of beer, compared with a water preload and no preload.

Regarding ratings of food liking, Caton et al. (2005) showed that pleasantness of food items did not differ between consumption of 24 g of alcohol and a soft drink. Yeomans (2010b) also failed to show any change in food pleasantness ratings between consumption of 12.5 g of alcohol and a fruit juice. However, Schrieks et al. (2015) showed that ratings of explicit liking of high-fat savory foods were significantly greater after 20 g of alcohol compared with an alcohol-free drink.

Desire to consume ratings between consumption of an alcoholic drink and an alcohol-free drink are also mixed. Hetherington et al. (2001) found no significant difference between an alcohol preload (24 g of alcohol) and an alcohol-free drink for desire-to-eat ratings. Caton et al. (2005) also found no difference in desire to consume ratings between an alcohol (24 g) and control condition. In contrast, Rose et al. (2015) showed that snack urge ratings (a composite score of expected liking, desire to consume, craving and difficulty to resist ratings) towards snack foods was greater after consumption of a 0.6 g/kg alcohol dose compared with a placebo. Westerterp-Plantenga and Verwegen (1999) found that desire to eat ratings were significantly *lower* after a 37.4 g alcohol preload of wine and a 34 g alcohol preload of beer, compared with a water preload and no preload.

Implicit measures of food reward have also been used to measure the effect of acute alcohol consumption. Karyadi and Cyders (2019) investigated whether food-related attentional biases can be increased by smelling alcohol odours, in the absence of alcohol consumption. This study showed that fixation duration towards food cues during a visual probe task, was greater following administration of an alcohol odour compared with a water odour. More recently, Monem and Fillmore (2019) examined the effect of alcohol intoxication on food-related attentional biases comparing attentional bias between an alcohol-free placebo and alcohol doses of 0.3 g/kg and 0.65 g/kg. Findings revealed that the magnitude of food-related attentional biases did not change between drink conditions, suggesting that in this study, acute alcohol consumption did not affect implicit food reward. Lastly, Adams and Wijk (2020) found that alcohol consumption did not alter the magnitude of attentional bias towards high-energy vs low-energy food cues when using a dose of 0.4 g/kg relative to a placebo.

Taken together, current findings suggest that the extent to which acute alcohol consumption affects explicit measures of food reward is mixed. Importantly, studies which have measured these components are heterogenous, with large variation in the type and dose of alcohol used, the alcohol-free comparator, the timings of alcohol consumption and absorption period, and sample size. In contrast to the meta-analysis by Kwok et al. (2019) which suggested that changes in eating behaviour are not observed at higher doses of alcohol, there is some evidence that alcohol dose may moderate these ratings, whereby *greater* alcohol doses produce *greater* increases in food reward. For example, Caton et al. (2004) demonstrated a dose-dependent response whereby hunger ratings increased when 32 g of alcohol were consumed, compared with 8 g. Similarly, the only study to have found an increase in snack urge ratings was Rose et al. (2015) who used a bodyweight dependent dose of alcohol (0.6 g/kg; mean weight in kg = 68.14, mean amount of alcohol consumed = 42 g). Although the weight of each individual participant is not reported, many participants in this study would likely have consumed an alcohol dose greater than the other reported studies which measured desire to eat, therefore a larger dose may be needed to observe such an effect. Interestingly, a dose-dependent effect was not seen within food-related attentional bias, as Monem and Fillmore (2019) showed no such effect across alcohol doses. One explanation for this null finding could be due to a lack of statistical

power. In their study, 23 participants completed all three drink conditions, which would provide statistical power to detect a medium-to-large effect. However as noted before, the relationship between food craving and food-related AB is small (Hardman et al., 2020). Therefore, Monem and Fillmore (2019) were likely underpowered to detect an effect of acute alcohol consumption on food-related AB.

Collectively, the current findings lack consistency and require future research to test for the possibility that acute alcohol consumption can increase food reward in a dose-dependent manner. Current evidence comes from highly heterogenous studies which differ on the types of alcohol, the alcohol-free comparator, the type of dose used (fixed dose vs body-weight dependent), the size of the dose, and sample size. Therefore, additional research which uses homogenous methodology, is needed. This includes using the same type of alcohol, alcohol-free comparator, type of dose and one which is adequately powered to detect likely effect sizes, in order to build on previous research which used an inadequate sample size (Adams & Wijk, 2020; Monem & Fillmore, 2019).

1.5.1.2. Acute alcohol consumption and episodic meal memories

Neurological evidence has demonstrated that acute alcohol consumption reduces activation within brain regions implicated in memory formation, such as the hippocampus (for review see White et al., 2000). Specifically, alcohol supresses the activation of pyramidal cells in region CA1 of the hippocampus (White & Best, 2000) - an area implicated in the process of forming new explicit memories (Zola-Morgan et al., 1986). Additional investigations have revealed that acute alcohol consumption also affects activation of other brain structures involved in memory encoding, including the parahippocampal gyrus and frontal lobes (Cabeza & Nyberg, 2000; Schacter & Wagner, 1999; Söderlund et al., 2007).

Studies in humans have demonstrated that acute alcohol administration produces impaired recall of episodic memories when information is presented when intoxicated (Bisby et al., 2010; Curran & Hildebrandt, 1999; Hashtroudi et al., 1984; Nilsson et al., 1989; Söderlund et al., 2005). Encoding deficits have been shown for both verbal free recall and recognition tasks (Williams & Rundell, 1984) as well as cued recall tasks (Söderlund et al., 2005). Evidence suggests that this effect

may be dose-dependent, as research has shown found an alcohol-induced impairment at a dose of 0.6 g/kg and 0.8 g/kg, but not at 0.4 g/kg, relative to placebo (Bisby et al., 2010).

Importantly, the order in which alcohol is consumed relative to presentation of stimuli can affect recall performance. For example, acute alcohol consumption has been shown to enhance recall of stimuli when it is consumed after the stimuli has been presented (Knowles & Duka, 2004; Parker et al., 1980; Weafer et al., 2016). Weafer et al. (2016) found that alcohol consumed after presentation of pictorial stimuli significantly improved recall compared with consumption of an alcohol-free placebo, suggesting that alcohol consumption can aid consolidation of recent memories and boost later recall. This effect, termed 'retrograde facilitation', is believed to result from alcohol's ability to enhance consolidation of stimuli. When alcohol is consumed after information is presented, memories formed prior to alcohol consumption are protected due to an alcohol-induced impairment in forming new memories, which reduces interference during this phase (Wixted, 2005).

As recall of episodic memories encoded in an intoxicated state is impaired relative to when encoding occurs whilst sober, meal memories may become disrupted when food is consumed in an intoxicated state due to impairments during the encoding phase. As reviewed in Section 1.2.8, when individuals are distracted away from a meal which they are eating, recall of the meal becomes impaired and subsequent food intake increases. Alcohol intoxication may produce a similar effect due to its ability to disrupt memory encoding, which may in part contribute to alcohol-induced overeating at a subsequent eating opportunity. Conversely, through alcohol's retrograde facilitation effect on memory formation, alcohol consumed after a meal may in fact boost meal memories relative to consumption of an alcohol-free drink which may then also affect subsequent intake. These possibilities are untested and no research to date has examined the effect of alcohol consumption on meal memories.

1.5.1.3. Alcohol's effect on impulsivity

Acute alcohol consumption has been shown to produce neurological change to brain regions implicated in impulsivity. Neurological evidence in humans has shown that acute administration of alcohol produces decreases in glucose metabolism within the prefrontal cortex (de

Wit et al., 1990; Volkow et al., 1990; Volkow et al., 2006) as well as areas which hold connectivity to prefrontal cortex regions, including the anterior cingulate cortex (Anderson et al., 2011; Marinkovic et al., 2012). Furthermore, relative to placebo, consumption of an alcohol dose of 0.6 g/kg decreases brain responses in the right fronto-temporal areas of the brain (Gan et al., 2014), a region implicated in response inhibition.

Studies have also investigated how acute alcohol consumption can affect performance on behavioural measures of impulsivity. Research which has investigated the effect of alcohol consumption on performance of a delay discounting task is mixed. Studies which have used an alcohol dose ranging from 0.4 – 0.8 g/kg, a fixed dose of 40 mg/dl and 80 mg/dl and a blood alcohol content of 0.08 have failed to show any change in performance on a delay discounting task (Adams et al., 2017; Bidwell et al., 2013; Richards et al., 1999; Wray et al., 2015). However, one study found that a dose of 0.8 g/kg, but not 0.4 g/kg, produced an increase in delay discounting relative to placebo (Reynolds et al., 2006b).

Several studies have shown that acute alcohol consumption impairs response inhibition at doses ranging from 0.4 – 0.65 g/kg (Abroms et al., 2003; Christiansen et al., 2016; de Wit et al., 2000; Fillmore & Vogel-Sprott, 2000; Marczinski & Fillmore, 2003; Marinkovic et al., 2012). Importantly, Weafer and Fillmore (2008) showed that after consumption of a 0.65 g/kg dose of alcohol, the magnitude of impaired inhibitory control (the difference after consumption of alcohol and a placebo) was positively correlated with *ad libitum* alcohol consumption in a separate test session, and explained 20% of the variance in alcohol consumption, suggesting that changes to inhibitory control is an important factor in alcohol-induced consumption of appetitive stimuli.

Only one study to date has investigated the extent to which alcohol-induced changes in inhibitory control affects eating behaviour. Christiansen et al. (2016) demonstrated that performance on the Stroop task after consumption of a 0.6 g/kg dose of alcohol (relative to an alcohol-free placebo) mediated the effect of drink condition on subsequent cookie intake, such that Stroop task completion time was significantly greater in the alcohol condition and that there was a positive association between completion time and the amount of cookies consumed.

1.5.1.4. A dual-process model of eating behaviour – the case for acute alcohol consumption.

Taking the findings from research examining food reward and inhibitory control, it is plausible that alcohol-induced food intake may occur through an interaction of changes to both top-down and bottom-up processes. This dual-process model has been previously investigated within the context of alcohol consumption and eating behaviour (Hofmann & Friese, 2008). In this study, participants completed an implicit association test (a measure of implicit attitudes towards candy) to assess baseline implicit attitudes, before consumption of a 0.4 g/kg dose of alcohol or a soft drink. After consumption of the drink, participants were given *ad libitum* access to the same food. Findings revealed that implicit attitudes before consumption of the test drink were a better predictor of food intake for participants who had consumed alcohol than those who had consumed the soft drink. The interpretation of these findings was that implicit attitudes determined candy consumption in the alcohol condition because in this condition, inhibitory control (top-down processes) would have been impaired, therefore allowing implicit attitudes (bottom-up processes) to drive eating behaviour.

Although Hofmann and Friese (2008) demonstrated the predictive power of implicit attitudes after alcohol consumption, no studies to date have measured how implicit attitudes towards food measured *after* alcohol consumption predict subsequent intake. As acute alcohol consumption may increase implicit positive attitudes towards food, subsequent food intake may be predicted by an interaction of alcohol-induced increases in motivational orientation of food and changes to inhibitory control.

1.5.1.5. Individual differences in susceptibility to alcohol-induced food intake

Particular characteristics may make some individuals more susceptible to alcohol-induced overeating than others. It may be possible that individuals who display poor baseline inhibition (in the absence of alcohol intoxication) are more susceptible to this type of overeating. In addition to previous evidence demonstrating that the interaction of impulsivity and appetitive responding to food predicts overeating and BMI (see Section 1.2.5), findings suggest that baseline inhibitory control affects weight change within the context of alcohol consumption (Kase et al., 2016). In this study,

Kase et al. measured the effect of alcohol consumption and behavioural impulsivity (using a Go/No-Go Task) within a weight loss treatment trial. Findings revealed that impulsivity and change in alcohol consumption interactively predicted weight loss, such that reductions in alcohol consumption predicted greater weight loss in individuals with high impulsivity - when impulsive individuals lowered their alcohol consumption, they experienced greater weight loss compared to individuals with lower levels of impulsivity. One interpretation of this finding is that higher baseline levels of impulsivity are exacerbated by alcohol-induced changes in inhibitory control, producing a compounded disruption to top-down processes, relative to individuals with lower levels of impulsivity. This in turn then leads to poorer inhibition of food intake which may be more desired due to alcohol-induced increases in appetitive responding to food. Therefore, in the context of a weight-loss trial, reduced alcohol consumption appears to be a more effective method of weight loss for individuals who display greater baseline impulsivity.

1.6. The effect of alcohol on weight-related measures

Given that acute alcohol consumption has been shown to consistently produce a caloric surplus relative to an alcohol-free beverage (Kwok et al., 2019), excessive alcohol consumption has been implicated as a risk factor for weight gain. However, evidence from meta-analyses (Sayon-Orea et al., 2011) and literature reviews (Traversy & Chaput, 2015) inclusive of both cross-sectional and longitudinal investigations of the effect of drinking behaviour on weight-related measures, suggest that this relationship is inconsistent. In the case of cross-sectional data, multiple studies have shown a lack of correspondence between alcohol intake and BMI in men, and a slight negative association in women (Bergmann et al., 2011; Colditz et al., 1991; Gruchow et al., 1985; Liangpunsakul, 2010; Rohrer et al., 2005; Skrzypczak et al., 2008; Wannamethee et al., 2004; Wannamethee et al., 2005; Williamson et al., 1987). Similarly, many studies have found no association between alcohol intake and changes in weight, BMI or other measures of adiposity, using a longitudinal design (Arabshahi et al., 2014; Halkjær et al., 2006; Holloway et al., 2011; Liu et al., 1994; Pajari et al., 2010; Sammel et al., 2003; Thomson et al., 2012; Tolstrup et al., 2008; Wang et al., 2010; Wannamethee et al., 2004).

The association between drinking behaviour and weight-related outcomes does not appear to be linear. Instead, a number of studies have shown that the relationship between drinking behaviour and weight-related outcomes is J-shaped, such that light-to-moderate drinkers display lower levels of adiposity compared with non-drinkers, whereas heavier drinkers display the greatest level (Arif & Rohrer, 2005; Duvigneaud et al., 2007; Lukasiewicz et al., 2005; Tolstrup et al., 2005; Wakabayashi, 2010; Wannamethee et al., 2005). Similarly, several studies have found a positive association between drinking behaviour and measures of adiposity in heavy drinkers (Halkjær et al., 2006; MacInnis et al., 2014; Rissanen et al., 1991; Sayon-Orea et al., 2011; Schütze et al., 2009; Wannamethee et al., 2004). Furthermore, a meta-analysis by Sayon-Orea et al. (2011) showed that a positive association exists between alcohol consumption and body weight for those who display heavy drinking patterns, but not for moderate or light alcohol drinkers (Sayon-Orea et al., 2011). Taken together, findings suggest that heavier drinking behaviours produce a greater risk for weight gain, whereas light-to-moderate levels appear not to pose a risk.

Another important distinction to make when examining this association is to distinguish how drinking behaviour is measured. Specifically, drinking behaviour which focuses on the frequency of consumption (i.e., how often alcohol is consumed) appears to be a less consistent predictor than other measures, such as drinking intensity (the average number of drinks consumed in a drinking episode) and drinking amount (the number of drinks consumed over a period of time). In support of this, three cross-sectional studies showed that drinking frequency negatively correlates with adiposity, whereas intensity positively correlates with weight-related outcomes (Breslow & Smothers, 2005; French et al., 2010; Tolstrup et al., 2005). Other studies have also demonstrated that drinking intensity and amount consumed is a significant predictor of adiposity. For example, Sa et al. (2019) showed that drinking intensity, but not drinking frequency, was positively associated with BMI. Wannamethee et al. (2005) found that men who drank ≥ 21 units per week showed higher levels of adiposity compared with non-drinkers and light drinks (< 21 units per week). Coulson et al. (2013) found that BMI, percent body fat and waist circumference were higher in individuals who consume five or more drinks per drinking day, compared with non-drinkers. Shelton and Knott (2014) measured energy intake

from alcohol on days where individuals had their highest levels of alcohol consumption. For the heaviest drinkers, risk of obesity was 70% higher compared with the lightest drinkers.

The type of alcohol regularly consumed may also moderate the association between drinking behaviour and adiposity. For example, Lukasiewicz et al. (2005) found that individuals who consumed less than one drink of wine per day had a lower waist-to-hip ratio (WHR) than non-drinkers or those consuming more, whereas a positive association between consumption of spirits and both BMI and WHR was found. Wannamethee et al. (2005) found a positive association between alcohol intake and BMI, WHR, waist circumference and percentage of body fat, however these associations were greatest in beer and spirit drinkers and weaker in wine drinkers. Vadstrup et al. (2003) found that the odds of having a high waist circumference increased for both men and women who consumed beer or spirits, but not wine. Differences in measures of adiposity between drink types has been suggested to occur due to an overall healthier diet of wine drinkers (Sánchez-Villegas et al., 2009). However, other reasons for a reduced association between drinking behaviour and adiposity in the case of wine may be explained by its components. One such component is resveratrol, which inhibits de novo lipogenesis and enhances the lipolytic effect of epinephrine, effects which may mimic caloric restriction (Fischer-Posovszky et al., 2010; Pedersen et al., 2008). Therefore, consumption of wine may reduce the risk of weight gain, relative to other types of alcohol.

1.6.1. Binge drinking and weight gain.

The association between heavy drinking patterns and adiposity has implicated binge drinking as a predictor of weight gain. The reason for this has been suggested to be, in part, related to acute effects of alcohol consumption. As reviewed, there is some (but mixed) evidence to suggest that the effect of alcohol consumption on eating behaviour is dose-dependent (Caton et al., 2004), whereby greater increases in caloric intake from alcohol as well as calories consumed from food may occur only after a certain level of intoxication. Therefore, it may be the case that when individuals engage in consumption of several drinks within the same drinking episode, not only may calories consumed

from alcohol be poorly compensated for, but individuals may be likely to overeat, creating a greater caloric surplus.

Alternatively, binge drinking may relate to adiposity due to its shared commonalities with other behaviours implicated in weight gain. Of note, binge drinking is associated with both binge eating behaviours (Harrell, Slane, & Klump 2009; Fischer & Smith, 2008; Krahn et al., 2005) and trait impulsivity (Benjamin & Wulfert, 2005; O'Halloran et al., 2018). These associated behaviours with binge drinking may mean that in some cases, binge drinking alone does not necessarily lead to weight gain. Instead, individuals who display binge drinking behaviours may be more at risk of weight gain because they are more likely to display other behaviours which cause weight gain, these factors may therefore potentially confound the link between binge drinking and weight-related outcomes.

1.6.2. Alcohol consumption as a predictor of weight gain in University students

The transition to University is a period where students experience significant environmental and lifestyle changes. Weight gain over the course of the first year of university has been reliably reported, with students on average gaining five pounds over this period of time (Vadeboncoeur et al., 2015; Vella-Zarb & Elgar, 2009). Previous research has identified several variables which predict weight change in first year undergraduates, including trait disinhibition and binge eating behaviours (Finlayson et al., 2012), decrease in physical activity (Butler et al., 2004), high levels of perceived stress (Serlachius et al., 2007), and high levels of unhealthy food consumption (Levitsky et al., 2004).

Alcohol consumption is another lifestyle factor which changes when transitioning to University. Drinking behaviour among undergraduate students is high, with approximately two-thirds of students from Ireland and the UK being classified as displaying harmful drinking (Davoren et al., 2016). Furthermore, alcohol consumption of undergraduates has consistently been shown to be greater than their non-attending counterparts (Johnston et al., 2015; Johnston et al., 2016; Kypri et al., 2005). Therefore, University students who engage in excessive alcohol consumption may be at risk of undesirable weight gain.

Several studies have investigated whether alcohol consumption predicts weight-related outcomes in undergraduate samples. However, findings are mixed, with some studies showing an effect (Adams & Rini, 2007; Bodenlos et al., 2015; Deforche et al., 2015; de Vos et al., 2015; Economos et al., 2008; Lloyd-Richardson et al., 2008; Zagorsky & Smith, 2011) and others failing to show an association (Deliens et al., 2013; Fazzino et al., 2019; Kasparek et al., 2008; Pliner & Saunders, 2008; Pope et al., 2017).

As with findings from the general population, the association between drinking behaviour and weight-related outcomes appears to vary based on the measure of alcohol consumption used. Of the five studies which did not find an association, three measured drinking frequency (Kasparek et al., 2008; Pliner & Saunders, 2008; Pope et al., 2017), one separately measured both drinking frequency and intensity (Deliens et al., 2013) and one measured number of heavy drinking episodes per month and mean total weekly drinks (Fazzino et al., 2019). Conversely, of the seven studies which did find an association, three measured the amount of alcohol typically consumed in a week (Bodenlos et al., 2015; de Vos et al., 2015; Economos et al., 2008), one measured alcohol consumption as a yes/no dichotomous variable (Adams & Rini, 2007), one categorised responses by non-drinkers, low-risk drinkers and moderate drinkers (Lloyd-Richardson et al., 2008), one study split respondents into heavy and non-heavy drinkers (heavy drinkers were defined as consuming six or more drinks four times over the past month) (Zagorsky & Smith, 2011) and one measured drinking frequency (Deforche et al., 2015). Therefore, although not entirely consistent, there appears to be some suggestion that drinking intensity and the amount of alcohol consumed is a better predictor than the frequency of drinking episodes.

1.6.3. Compensatory behaviours of alcohol consumption

An additional reason why previous studies have failed to show an association between drinking behaviour and weight-related outcomes may be the result of not controlling for confounding variables, such as compensatory behaviours. One example of a compensatory behaviour is the initiation of physical activity in response to alcohol consumption. Data from systematic reviews have demonstrated a consistent positive association between physical activity and alcohol use in youth

samples, university students, and the general population (Dodge et al., 2017; Piazza-Gardner & Barry, 2012). One suggestion for why physical activity positively correlates with alcohol consumption is that health motivations (in particular the desire to combat against an alcohol-induced caloric surplus) may partially explain this association. In support of this, evidence from Dodge and Clarke (2018) showed that a positive association between heavy episodic drinking and vigorous physical activity was mediated by health motives, suggesting that levels of physical activity can be affected by drinking behaviour, due to health concerns generated by alcohol consumption.

In addition to physical activity, other forms of caloric compensation may be initiated in response to alcohol consumption. 'Drunkorexia' refers to compensatory behaviours in association with alcohol consumption with the goal to reduce caloric consumption and/or enhance the effects of alcohol through means of caloric restriction, purging, as well as physical activity (Bryant et al., 2012). In the case of caloric restriction, evidence suggests that the percentage of university students who have changed or restricted their eating behaviour prior to alcohol consumption ranges from 14% - 46% (Burke et al., 2010; Giles et al., 2009; Roosen & Mills, 2015). Findings suggest that some alcohol-related compensatory behaviours including physical activity and dietary restraint occur similarly in both males and females (Gorrell et al., 2019; Peralta & Barr, 2017; Rahal et al., 2012). However, other types (such as bulimic-type behaviours) appear to be more prevalent in females (Gorrell et al., 2019).

1.7. Summary and Thesis Aims

Drawing on contemporary models of appetite control, this thesis aims to further the understanding of key psychological mechanisms which may contribute towards alcohol-induced changes in eating behaviour. The thesis will focus on two components of eating behaviour - cognitive control of eating and reward processes - which to date, have received limited empirical interest in the context of alcohol-induced eating. Regarding cognitive processes, the ability of acute alcohol consumption to disrupt episodic meal memories is a promising, yet understudied, factor which could affect alcohol-induced food intake and will be a key focus of this thesis. Reward processes will also be investigated, as current evidence is mixed as to whether acute alcohol consumption can increase

components of food reward using both explicit and implicit measures. More specifically, the thesis will address these inconsistencies by investigating whether the effect of acute alcohol consumption on food reward is dose dependent – this effect will be measured at a dose of 0.3 g/kg and 0.6 g/kg, relative to a placebo. Furthermore, the thesis will also use larger sample sizes than previous research to ensure that a small effect of acute alcohol consumption on food reward can be detected. The thesis will also address previous methodological issues relating to attentional bias by comparing food images with non-food control images, rather than by comparing between different types of food, as done by Adams and Wijk (2020).

The thesis will also bring together reward and cognitive processes by incorporating a dual-process perspective of eating behaviour in the context of acute alcohol consumption. This will be achieved by examining whether motor trait impulsivity (i.e. indicating a weaker reflective/top-down system) and alcohol-induced changes in food reward (i.e. bottom-up system) interactively predict changes in food intake. Currently, no studies have examined the predictive value of the interaction between top-down and bottom-up processes in the context of alcohol-induced food intake. This dual-process approach will also be tested by examining whether alcohol consumption predicts weight-related outcomes over time and whether motor trait impulsivity moderates this effect. Therefore, the overarching aim of this thesis is to examine the role of cognitive and reward-related processes associated with both acute and long-term alcohol consumption on eating behaviour and weight-related outcomes.

Aim 1: To measure the influence of acute alcohol consumption on cognitive processes implicated in eating behaviour.

Chapter 3 consists of two laboratory studies; both investigate whether acute alcohol consumption can affect episodic memories related to a recently consumed meal. Study 1 examines how consumption of an alcoholic drink (dose of 0.5 g/kg) prior to a lunch meal affects subsequent recall of that lunch meal and to see whether an impairment of meal memory can affect subsequent food intake, relative to consumption of an alcohol-free placebo. This study tested the hypothesis that consuming an alcoholic drink would impair meal memory of the lunch meal, and that this impairment

would mediate the effect of drink condition on subsequent food intake. Study 2 examines whether the order in which alcohol is consumed, relative to a lunch meal, affects subsequent meal memory recall and food intake. Here, the retrograde facilitation effect (Wixted, 2005) is tested to see whether consuming alcohol *after* a lunch meal produces greater meal memory and lesser food intake, relative to when an alcoholic drink or a soft drink is consumed *before* a lunch meal (both alcoholic drinks have a dose of 0.6 g/kg).

Aim 2: To measure the influence of acute alcohol consumption on both implicit and explicit measures of food reward.

Chapter 4 consists of a further two laboratory studies, both of which focus on the effect of acute alcohol consumption on food reward. In Study 3, the effect of alcohol on self-report measures of food reward and food-related attentional bias is measured and compared between consumption of a 0.3 g/kg dose of alcohol and an alcohol-free placebo, in order to test whether acute alcohol consumption can enhance both explicit and implicit measures of food reward and increase *ad libitum* food intake. Study 4 builds upon Study 3, by investigating whether alcohol-induced enhancements of food reward are present at a higher dose (0.6 g/kg) relative to a placebo by again measuring self-reported components of food reward and food-related attentional bias. Study 4 also investigated whether the same alcohol dose can enhance alcohol-related reward relative to placebo, through self-report measures and an alcohol-related attentional bias.

Aim 3: To investigate whether a dual-process model of eating behaviour can account for alcohol-induced changes in acute eating behaviour and longer-term BMI change.

The final aim of the thesis is to examine whether a dual-process model of eating behaviour can explain alcohol-induced changes in food intake and BMI. The laboratory study outlined above (Study 4) additionally examined whether trait motor impulsivity (a top-down process) and the difference in attentional bias towards food cues (a bottom-up process) after consumption of an alcoholic drink (0.6 g/kg) and a placebo, predict difference in food intake between the two drink conditions. Lastly, Chapter 5 (Study 5) investigates whether longer-term drinking behaviour can

predict BMI. Using a longitudinal design, the drinking behaviour and BMI of first-year undergraduates were recorded at baseline, six months and 12 months. This study aimed to test whether changes in drinking behaviour predict BMI by ensuring that established confounding variables (including physical activity and compensatory eating in response to alcohol consumption) were controlled for. The study also examined the effect of drinking frequency and drinking intensity on BMI, separately. A final aim of this study was to investigate whether trait motor impulsivity and drinking behaviour interactively predict changes in BMI.

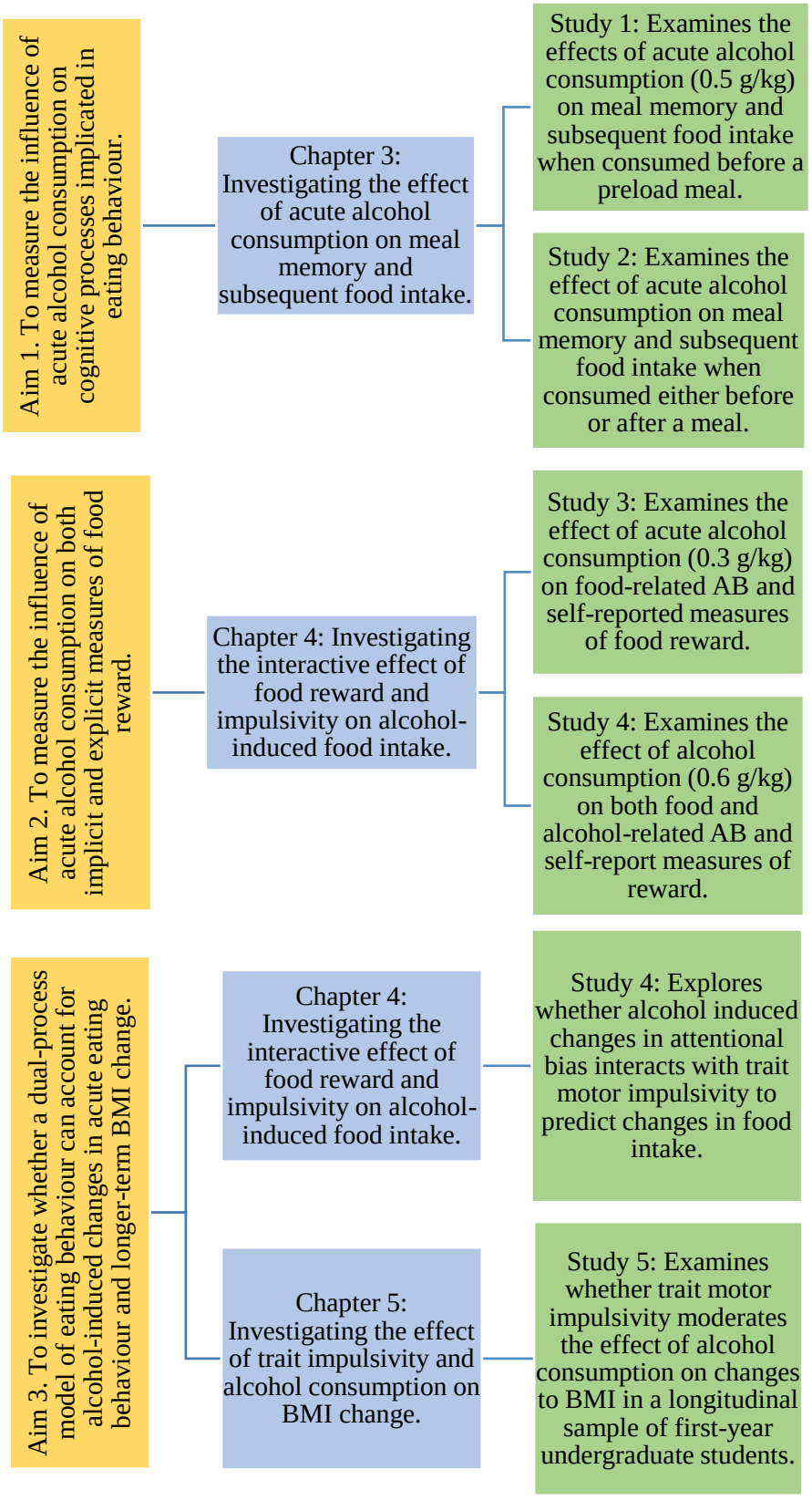

Figure 1.1. Schematic overview of thesis structure. Thesis aims are in gold, chapter headings are in blue. Chapter aims are in the green boxes.

Chapter 2: General Methods

Numerous methods were used throughout the thesis, this section provides a list of each method, which describes the method and provides rationale for their use.

2.1. Questionnaire Measures

2.1.1. Alcohol Use Disorders Identification Test

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) was used in Studies 1-4 to characterize the sample regarding hazardous and harmful drinking behaviour. The AUDIT consists of ten questions which capture consumption of alcohol (i.e., frequency and quantity patterns), alcohol dependency, and harmful alcohol use (including concern of others). Each question is scored from 0-4, creating a maximum possible score of 40. A score of ≥ 8 is considered the threshold for harmful or hazardous drinking, and a score of ≥ 20 is indicative of alcohol dependence (Babor et al., 2001). The AUDIT has been shown to have a high level of internal consistency across many different settings and samples, ranging between 0.75 and 0.97 (Reinert & Allen, 2007). Similarly, test-retest reliability of the AUDIT has been shown to be good across samples (Dybek et al., 2006; Selin, 2003).

2.1.2. Timeline Followback

The 7-day Timeline Followback (TLFB; Sobell & Sobell, 1992) was completed in Studies 1-4 to measure how many UK units of alcohol (1 UK unit = 8 grams of alcohol) participants had consumed in the last seven days. This measure is widely used to capture retrospective estimations of alcohol consumption and can range in length from one week to twelve months (Sobell & Sobell, 1992), although greater durations of the TLFB may produce greater inaccuracies (Hoeppner et al., 2010). Therefore, studies in the current thesis only recorded retrospective unit consumption over the past seven days. The TLFB has good reliability, and has been shown to have high test-retest reliability among both individuals with alcohol dependency and social drinkers (Hoeppner et al., 2010).

2.1.3. Dutch Eating Behaviour Questionnaire

The Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien et al., 1986) is a validated 33-item questionnaire which measures eating styles associated with being overweight and consists of three subscales: restraint, emotional eating, and external eating. The restraint sub-scale of the DEBQ has high test-retest and internal consistency (Allison et al., 1992) The DEBQ was adminstered in Studies 1-4 to characterize the sample in terms of their eating behaviour styles.

In Studies 3 and 4, restraint scores of the DEBQ were included as a moderator between drink condition and food intake. This was used as a moderator because individuals with dietary restraint may be more susceptible to alcohol-induced overeating, possibly due to a reduction in the ability to maintain restrained eating behaviours, resulting in a temporary change to dietary intentions (Caton, Nolan, & Hetherington, 2015). This effect was first studied by Polivy and Herman (1976a; 1976b) who found that when restrained eaters (as measured using the restraint scale; Herman & Polivy, 1975) were aware of the presence of alcohol, their eating behaviour became disinhibited. Whereas, when restrained eaters were unaware of the presence of alcohol, food intake was suppressed (relative to unrestrained individuals), suggesting that alcohol-related expectancy effects may contribute towards disinhibited eating in restrained individuals. However, subsequent research has been unable to demonstrate that restrained eaters are more susceptible to alcohol-induced increases in food intake (Christiansen et al., 2016; Poppitt et al., 1996; Yeomans, 2010b; Yeomans, Hails, & Nesic, 1999), even when they are made aware of the presence of alcohol (Ouwens et al., 2003). This discrepancy in findings may have occurred due to differences in how dietary restraint was measured, with suggestions that the restraint scale captures unsuccessful dieting, whereas the DEBQ captures successful dieting (Laessle et al., 1989). Therefore, DEBQ restraint scores were used to further investigative this possibility.

2.1.4. Snack Urge Ratings

A snack urge ratings scale (Hardman et al., 2015) was administered in Studies 1-4 in order to investigate how time and the type of drink consumed can affect state-like self-report ratings relating to food reward. The scale ratings were obtained using 100- mm visual analogue scales from the of four

items - “How much do you expect to like this food?”, “How strong is your desire to eat this food right now?”, “How strong is your craving for this food right now?”, and “How difficult is it to resist eating this food right now?” The anchor points for all scales were “Not at all” and “Extremely”. Ratings were asked in relation to the test foods which participants would consume in the *ad libitum* taste test for that study. Scores were totalled and combined as a composite score in order to capture overall food reward ratings. These four items were used because of previous suggestions that food reward consists of both ‘liking’ and ‘wanting’ of a food, and whereby liking and desire-to-eat a food are related (Rogers & Hardman, 2015). Therefore, instead of providing a measure of food reward which captures one component (e.g., desire to eat), this measure captures total food reward.

2.1.5. Appetite Ratings

Self-report appetite ratings were measured in Studies 1-4 in order to measure how time and the type of drink can affect changes of ratings related to appetite. The measure consisted of two ratings using 100- mm visual analogue scales, the ratings were: “I feel hungry” and “My stomach feels full” with anchors “Not at all” and “Extremely”. These scores were combined (hunger added to the inverse score of fullness) and reported as a single appetite rating (maximum score of 200) as hunger and fullness ratings are negatively correlated with one another (Rogers & Hardman, 2015).

2.1.6. Barratt Impulsiveness Scale

The Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995) is a questionnaire designed to measure personality characteristics relating to impulsivity. Principal components analysis of BIS-11 scores have identified three second order factors of impulsivity (Patton et al., 1995) – these are: non-planning (lack of future thinking), motor (acting without thinking), and attentional impulsivity (inability to focus attention). The BIS-11 has good levels of internal consistency (Vasconcelos et al., 2012) and test-retest reliability at one month (Stanford et al., 2009).

Evidence suggests that impulsivity as measured by the BIS-11, is linked with overeating and BMI across different types of measures. For example, motor impulsivity has been shown to be higher in groups with binge-eating behaviours compared with controls (Nasser et al., 2004; Rosval et al., 2006). Lyke and Spinella (2004) showed that the attentional and motor impulsivity subscales were

positively correlated with the disinhibited eating subscale of the Three-Factor Eating Questionnaire (Stunkard & Messick, 1985). An additional two studies have shown that the subscale of motor impulsivity, but no other BIS-11 subscales or total score, were positively associated with BMI (Price et al., 2015; Van Koningsbruggen et al., 2013). Furthermore, motor trait impulsivity scores have been shown to interact with food-related attentional bias to predict weight gain (Meule & Platte, 2016). Therefore, because the motor impulsivity subscale appears to be particularly linked with food intake and BMI, scores on this subscale were included as a moderator between the effect of drink type and food intake in Study 4, and a moderator between drinking behaviour and BMI in Study 5.

2.1.7. Biphasic alcohol effects scale

The Biphasic Alcohol Effects Scale (BAES; Martin et al., 1993) is a validated 14-item scale which is comprised of two 7-seven item sub-scale, measuring the sedative and stimulating effects of alcohol. Participants were required to rate the extent to which they are experiencing both sedative (e.g., down, inactive) and stimulatory feelings (e.g., elated, energized) at the present moment on a 10-point scale. Anchored scores are 'Not at all' and 'Extremely'. In Study 1, BAES scores were measured at baseline, after consumption of the test drink, after consumption of the lunch meal and after consumption of the taste test in order to capture changes in feelings of sedation and stimulation across the study session, as an ascending blood alcohol concentration has been shown to produce stimulating effects, whereas descending blood alcohol concentration produces sedative effects (Tucker et al., 1982; Hendler et al., 2013). Internal reliability is high for both subscales, with Cronbach's alpha ranging from 0.85 to 0.94 (Martin et al., 1993) and findings have supported a factor structure in line with a sedation/stimulation distinction during the ascending and descending limbs of breath alcohol concentration (Rueger et al., 2009).

2.1.8. Alcohol Urge Questionnaire

The Alcohol Urge Questionnaire (AUQ; Bohn et al., 1995) is a validated measure of drinking urge across three domains: desire for alcohol; expectation of positive effect from drinking; and inability to avoid drinking if alcohol was available. Items are responded to on a scale from 1 to 7 with high scores being indicative of greater alcohol urge. The AUQ has been used in laboratory studies

assessing alcohol craving (Hutchinson et al., 2003; MacKillop & Lisman, 2005) and is sensitive to alcohol-induced changes in alcohol craving (O'Malley et al., 1997). The AUQ has been shown to have good internal consistency as a single factor measure of alcohol urge (Bohn et al., 1995). Participants completed this measure in Study 4 in order to capture changes in urge ratings before and after consumption of their test drink.

2.1.9. Subjective Intoxication Scale

The Subjective intoxication scales (SIS; Duka et al., 1998) measures subjective feelings of being 'lightheaded', 'irritable', 'stimulated', 'alert', 'relaxed', and 'contented' on a 5-point Likert scale. In Study 4, these ratings were taken at baseline and after consumption of the test drink in order to measure the effect of the test drink on these ratings. This measure has been used in several studies to capture alcohol-induced changes of subjective feelings (e.g., Baines et al., 2019; Christiansen et al., 2017; Duka et al., 1998).

2.1.10. Compensatory Eating and Behaviours in Response to Alcohol Consumption Scale

The Compensatory Eating and Behaviours in Response to Alcohol Consumption Scale (CEBRACS; Rahal et al., 2012) is a measure which assesses eating behaviour in relation to alcohol consumption before during and after alcohol consumption. The CEBRACS is a validated measure consisting of 21 items. A factor analysis has identified four separate factors for this scale: alcohol effects (restricting food intake to enhance the effect of alcohol), bulimia (reflecting bulimic-like behaviours in response to alcohol consumption), dietary restraint and exercise, and restriction (skipping meal or eating less in a day). Internal reliability scores for each sub-scale are good, ranging from 0.79 to 0.95 (Rahal et al., 2012). The CEBRACS has been shown to produce good internal reliability and construct validity cross-culturally too, for example in an Italian sample (Pinna et al., 2015). Study 5 used the CEBRACS to capture caloric compensatory behaviour before, during, and after consuming alcohol, over the past month in order to control for such compensatory behaviours. All items were summed to produce a total score.

2.1.11. International Physical Activity Questionnaire

The short-form International Physical Activity Questionnaire 7-day short form (IPAQ; Craig et al., 2003) was used in Study 5 to capture levels of physical activity. Participants were asked to report both the frequency (number of days in the last 7 days) and amount of time which they engaged in three levels of exercise: walking, moderate physical activity and vigorous physical activity in a day. The metabolic equivalent of task (MET) of each PA intensity level was multiplied by the duration and the frequency of the PA and was expressed as MET-minutes per week (MET-min/wk). Each MET-min/wk was then summed across the three levels to produce a measure of total physical activity.

A systematic review by Lee et al. (2011) has shown that the correlation between total physical activity measured by the short form IPAQ and objective measures of physical activity range from 0.09 to 0.39 (below the minimal acceptable standard). Whereas correlations for vigorous and moderate physical activity with their respective objective standards were above the minimal standard in several studies.

2.1.12. Food frequency questionnaire:

Study 5 included the food frequency questionnaire to capture dietary behaviour. The measure was a shortened version of a previously validated snack intake measure (Inchley et al., 2001) and consists of four questions which measured how often respondents consume savoury snack foods, sweet snack foods, convenience foods, and fast foods/take away foods. Eight options were presented and ranged from ‘Never or less than once a month’ to ‘More than 3 times a day, every day’. The score across the four items were summed as a total score (out of 32). The food frequency questionnaire focuses only on consumption of palatable foods as previous research has demonstrated increased preferences and intake of palatable foods (Caton et al., 2004; Schrieks et al., 2015; Christiansen et al., 2016). This score was included to characterise the sample in terms of level of unhealthy food consumption.

2.1.13. Body mass index

In all studies, body mass index (BMI) was calculated. For Studies 1-4, BMI was calculated in order to characterize the sample and was calculated using a stadiometer and weighing scales and was measured by the experimenter. In Study 5, BMI was used as a dependent variable and was calculated using self-reported height and weight. Self-report height and weight measurements were used for greater practical ease of obtaining this data, relative to measuring height and weight in a laboratory. In order to ensure that self-reported height and weight were of a good standard, responses outside of a biological plausible range (1.22 – 2.13 m for height and 34 – 227 kg for weight) were used as cut-offs, as has been done in previous research (Kersbergen & Robinson, 2019; Noël et al., 2010). However, this does not mitigate the issue of underreporting of weight and overreporting of height, which has been previously documented (Elgar & Stewart, 2008; Gorber et al., 2007).

2.2. Behavioural Measures

2.2.1. Memory Measures

Studies 1 and 2 included measures of meal memory and general memory recall in order to measure alcohol-induced changes to memory performance. Study 1 included previously used measures of meal memory recall. Firstly, participants were asked on a 100-mm Visual Analogue Scale 'How vividly can you remember the lunch meal you ate earlier?' Anchored scores were 'Not At All' and 'Extremely'. This measure of meal vividness has been used in previous investigations of meal memory (Higgs & Donohoe, 2011; Whitelock et al., 2018a). Participants also completed a free recall task of their lunch meal whereby participants were required to recall the nine food items they consumed during the lunch meal in no specific order. Participants also completed this same task but were asked to recall the specific order in which the nine food items were presented. These two measures were the same as measures used in a previous investigation of meal memory (Oldham-Cooper et al., 2011). Participants also completed a general memory measure whereby participants were shown a wordlist consisting of six capital cities and six countries to memorise, this measure was taken from the same previous study as the meal memory recall tasks (Oldham-Cooper et al., 2011).

Study 2 included the same meal vividness rating as in Study 1, but also included different measures of meal memory. Firstly, memory for satiety was measured, as has been done in previous investigations of meal memory (Whitelock et al., 2018a). For this, participants completed a computerised task in which they were asked to select the portion size of 18 meal foods to indicate the amount of food that would be required to produce the sensation of fullness that they experienced after lunch, this was adapted from Brunstrom et al. (2008). Meal memory was also measured using a visual memory task as has been done in previous research (Whitelock et al., 2018a) . For this, participants were presented with a large bowl of pasta salad (twice the amount of the same pasta salad they were served for lunch). Participants were asked to self-serve the amount of food which they believe they were served earlier for lunch, from the bowl onto a plate. The outcome measure was the difference between the amount of pasta self-served and the actual amount of pasta served at lunch, converted into an error percentage.

For the measure of general memory recall, a surprise free recall based on a previously seen picture presentation was used. Participants were given 5 minutes to recall as many of the picture text labels as they could remember from a previously shown presentation of image. Participants were told to recall the exact text of each label in any order they wished, and to avoid recalling any related words or synonyms. A response was marked as correct if it was the same text, with the exception of pluralising the word or recalling the text label correctly, but with incorrect spelling. The dependent variable was the number of text labels correctly recalled for each presentation set.

2.2.2. Attentional Bias

Studies 3 and 4 both included an attentional bias task. This task was included in order to measure implicit food reward. Food-related attentional bias has been shown to be positively related to self-report measures of food reward (Hardman et al., 2020). In Study 3, a visual dot probe task with concurrent eye tracking was used as the measure of attentional bias. Specifically, fixation duration towards target stimuli from concurrent eye-tracking was the outcome measure as this has greater internal reliability as compared with reaction time assessments when measuring food-related AB

using the visual probe task (van Ens et al., 2019). Food-related stimuli were separated into palatable and unpalatable food images. Palatable food images were of tortilla chips and chocolate chip cookies as these foods are high in fat and/or sugar. Comparatively, unpalatable foods were boiled potatoes and whole meal bread. These foods were chosen because they are relatively low in fat and sugar, and because they share some of the same visual characteristics (i.e., colour) as the palatable food images. Control (non-food) images were also included, these were drink coasters and leaves in order to match the other stimuli on visual characteristics.

In Study 4, a modified stroop task was used as a measure of attentional bias for both food and alcohol-related stimuli. For this task, each image was surrounded by a coloured border, participants were required to respond to the border colour as quick as possible, using a key response. This task was used due to concerns regarding oculomotor impairments following alcohol consumption at high doses (Abroms et al., 2006; Moser et al., 1998; Rohrbaugh et al., 1988). An AB task which uses ocular behaviour (i.e., eye movements) as its outcome measure may mask an effect of AB when using higher doses of alcohol. Furthermore, reaction time measurement in the modified stroop task has been shown to produce acceptable levels of internal reliability (Ataya et al., 2012). The same palatable food and control images were used (unpalatable food images were not included in Study 4) for the food-related attentional bias task. The alcohol-related attentional bias task was included as an appetitive comparator to food, and consisted of alcoholic drinks and stationery (both were matched on visual characteristics).

2.2.3. Taste test

In studies 1-4, *ad libitum* food intake was measured using a bogus taste test. The taste-test has been shown to be a valid measure of food intake (Robinson et al., 2017). In studies 1 and 2, the test meal consisted of a 200 g serving of Maryland chocolate chip cookies (487 kcal/100g) and a 250-gram serving of water (studies 1 and 2). Cookies were chosen as the test food because although studies have shown acute alcohol consumption to increase liking of savoury but not sweet foods (Schrieks et al., 2015) and that higher doses of alcohol can increase food intake of crisps, relative to lower doses (Caton et al., 2004), findings have also shown acute alcohol consumption to increase

consumption of cookies relative to placebo (Christiansen et al., 2016). Furthermore, the mechanism investigated in studies 1 and 2 is episodic meal memory. Disruption to episodic meal memory has been shown to increase cookie intake (Oldham-Cooper et al., 2011), and do not appear to be specific to savoury or sweet foods, therefore cookies were used as the test food in those studies. In Studies 3 and 4, the test meal consisted of 200 g of cookies and 200 g of tortilla chips with a 400 g serving of water.

2.3. Drink administration

Studies 1-4 included the administration of alcohol with either a placebo or a soft drink comparator. The dose of alcohol across these studies was weight dependent, as body weight is a significant determinant in the metabolism of alcohol (Cederbaum, 2012). The alcoholic drink used for all of these studies contained vodka (Smirnoff Red, 37.5% ABV) up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal) and was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. This vodka and lemonade mix was chosen as the alcoholic drink because of its greater percentage of alcohol content relative to other alcoholic drinks (e.g., beer), resulting in consumption of a smaller volume of liquid. In studies 1, 3 and 4 an alcohol-free placebo comparator was used. The placebo drink consisted of diet lemonade only; a vodka mist was sprayed on the surface of the drink to create the impression that it contained alcohol, as has been used in previous research (e.g., Christiansen et al., 2016). This alcoholic drink/placebo comparison was implemented in order to control for expectancy effects, as changes to appetite-related behaviours can be affected by alcohol-related expectancy effects alone (Christiansen et al., 2013; Polivy & Herman, 1976a, 1976b; Yeomans & Phillips, 2002). In Study 2, an alcohol/soft drink comparison was used, whereby participants were aware that the drink contained no alcohol when consuming the soft drink. This was done in order to observe behaviour in a more naturalistic context and because findings suggest that the observed effect in Study 2 is not affected by expectancy effects (Hull & Bond, 1986).

The dose of alcohol varied between studies, based on theoretical reasoning. In Study 1, we used a dose of 0.5 g/kg as similar doses have been shown to impair forms of memory recall (Bisby et al., 2010). In Study 2, this was raised to 0.6 g/kg, due to the null finding of memory recall in Study 1,

and based on findings that alcohols' memory impairment increases in a dose-dependent manner (Bisby et al., 2010). In Study 3, a dose of 0.3 g/kg was used because previous research has shown that this dose enhances attentional bias towards appetitive stimuli (Duka & Townshend, 2004; Schoenmakers et al., 2008). However, Study 4 used a dose of 0.6 g/kg due to a failure to detect an effect of attentional bias between drink conditions in Study 3, and because self-report measures of appetitive motivational states become increased at this dose 0.6 g/kg (Duka et al., 1999; Rose et al., 2015; Rose & Duka, 2006).

The amount of time participants had to consume the test drink varied between 10-15 minutes across studies. In Studies 1 and 3, participants were given 10 minutes to consume their test drink, whereas participants in Studies 2 and 4 had 15 minutes, and the drink was served in three separate portions in 5-minute intervals. This was done because Studies 2 and 4 used the larger 0.6 g/kg dose, therefore resulting in a greater volume of liquid to consume. The absorption period varied across studies too. In Studies 1, 2 and 3, a 10-minute absorption was implemented, whereas the absorption period in Study 4 was 20 minutes. This was altered because of the large volume of liquid consumed in Study 4, which was shortly followed by *ad libitum* consumption of the test food. It was anticipated that a 10-minute absorption period would affect satiety, potentially lowering food intake.

Prior to the test session, participants were instructed to consume a light meal, not high in fat (e.g., a sandwich) approximately an hour before the beginning of the session. This was done in order to ensure that participants did not consume alcohol on an empty stomach (which may otherwise produce nausea and vomiting) and also to standardize alcohol metabolism and appetite. This was checked at the beginning of each session by asking participants to report what and when they had last eaten. Sessions were rescheduled if participants had not complied with this instruction. Participants were not required to consume the exact same lunch across conditions in studies which used a within-subjects design.

Chapter 3: Investigating the effect of acute alcohol consumption on meal memory and subsequent food intake

3.1. Overview

Current understanding of the cognitive factors which underpin the effect of acute alcohol consumption on food intake is limited, despite their importance in determining appetite control (Higgs & Spetter, 2018). Across two studies, Chapter 2 examined the effect of alcohol intoxication on recall of a recently consumed meal. These two studies are the first to investigate how acute alcohol consumption can disrupt recall of food-specific episodic memories and whether any change to this cognitive performance can affect subsequent food intake.

The study reported in this chapter is currently under review as: Gough, T., Christiansen, P., Rose, A., & Hardman, C.A (under review). The effect of acute alcohol consumption on meal memory and subsequent food intake: Two laboratory experiments

3.2. Abstract

Altering the quality of episodic meal memories has been shown to affect subsequent food intake. Acute alcohol consumption disrupts memory formation and produces short-term overeating. In two studies, it was investigated whether alcohol consumption can affect meal-related memories and later food intake. Study 1 (N = 60, 50% male) investigated how consumption of an alcoholic drink (0.5 g/kg) prior to consumption of a lunch meal affected meal memory of that lunch, and later food intake, compared with a placebo-alcohol. Findings revealed that alcohol consumption did not impair meal memory, but did not affect subsequent food intake. Study 2 (N = 72, 50% male) investigated whether, due to alcohol's retrograde facilitation effect (the enhancement of recall due to reduced interference at the point of exposure), consuming alcohol *after* consumption of a lunch meal could enhance meal memory, compared with when consumed before a lunch meal (both a dosage of 0.6 g/kg), and compared with consumption of a soft drink. Contrary to prediction, alcohol consumed after a lunch meal did not significantly increase meal memory. But, as in Study 1, certain types of meal

memory were impaired when alcohol was consumed before the meal, compared with consumption of a soft drink. Subsequent food intake did not differ between conditions. Taken together, findings from both studies show that alcohol intoxication can impair some forms of meal memory recall, likely due to disruption of memory formation during the encoding phase. However, there was no evidence that this impairment contributes towards alcohol-induced overeating.

3.3. Introduction

A multitude of cognitive processes have been identified as factors which influence eating behaviour (Higgs & Spetter, 2018). Such factors include attention and memory for recent eating in determining food intake. A large body of research has demonstrated that impairments to episodic memories relating to recently consumed food can alter subsequent food intake. For example, animal research has demonstrated that lesions to the hippocampal region results in hyperphagia and weight gain (Clifton et al., 1998; Davidson et al., 2005). Furthermore, evidence from amnesic patients has demonstrated that individuals who have an impaired ability of reporting memories for recent eating also display evidence of overeating (Higgs et al., 2008b).

Manipulating the quality of episodic meal-related memories also affects subsequent food intake. This has been investigated by either enhancing or impairing the quality of a meal memory. Research has shown that cueing memory for a recently consumed meal reduces subsequent food intake, relative to no cue (Higgs, 2002; Higgs et al., 2008a). Similarly, many studies have investigated the effect of enhancing the level of attention placed towards a meal (specifically relating to the sensory properties of the food) on subsequent food intake, which has been suggested to increase meal memory. Findings are mixed, with some experiments showing that this increase in focused attention leads to a reduction in later food intake in both samples exclusively of women (Higgs & Donohoe, 2011; Robinson et al., 2014b) and a mixed gender sample (Seguias & Tapper, 2018). Other studies, however, have failed to show this same reduction in a mixed gender sample (Whitelock et al., 2018a), a male-only sample (Whitelock et al., 2019) and most recently a female-only sample (Tapper & Seguias, 2020).

Other research has focused on the effect of impairing memories of a recently consumed meal on subsequent food intake. This has been investigated by taking attention away from a meal whilst eating by using distractors such as television viewing (Higgs & Woodward, 2009; Mittal et al., 2011) and playing computer games (Oldham-Cooper et al., 2011). These studies have demonstrated that distracted participants display poorer levels of recall for meal memory, and greater subsequent food intake, compared with participants who eat in the absence of a distractor. This impairment of episodic meal memory is argued to be due to disruption during the encoding phase of memory formation.

Acute consumption of alcohol has been shown to impair processes of episodic memory, resulting in impaired delayed recall of stimuli when exposure or learning occurs shortly after alcohol consumption (Hashtroudi et al., 1984; Nilsson et al., 1989; Söderlund et al., 2005). This is believed to occur due to alcohol-induced disruptions to activity in the CA1 region of the hippocampus (White, Matthews & Best, 2000; Zola-Morgan et al., 1986). To date, no studies have investigated how acute alcohol consumption can impair recall of recently consumed food.

Acute alcohol consumption has also been shown to increase short-term levels of food intake, relative to consumption of alcohol-free drinks (Caton et al., 2004; Caton et al., 2005; Kwok et al., 2019; Yeomans, 2010a). Several mechanisms are likely to contribute to alcohol's effect on increased intake, such as impairment of inhibitory control (Christiansen et al., 2016) and enhancing the reward value of certain foods (Rose et al., 2015). However, a currently unexplored, but potentially important mechanism of this increased food intake may come from disruptions to meal memory if an alcoholic drink is consumed before consumption of food.

Sex differences in alcohol-induced changes to meal memory and food intake may also exist, as alcohol is metabolised differently between men and women (Ammon et al., 1996; Thomasson et al., 1995), resulting in a greater blood alcohol concentration in women (Mumenthaler et al., 1999). As the effect of acute alcohol consumption on food intake may varying according to dose (Caton et al., 2004), it is possible that women may display a greater effect of food intake after consumption of an alcoholic drink, relative to men. Findings have also shown that acute alcohol consumption can affect

memory performance to a greater extent in women than men (Jones & Jones, 2014; Niaura et al .,1987), therefore meal memory impairments may differ between men and women.

3.4. Study 1

3.4.1. Overview

In Study 1, participants either 1) consumed a pre-load meal after consuming an alcoholic drink, or 2) consumed a pre-load meal, after consuming a placebo-alcohol drink. After a delay of 30 minutes, all participants were given *ad libitum* access to chocolate chip cookies and recalled details of the pre-load meal. We hypothesised that participants who consumed an alcoholic drink would show greater impairment of meal memory and greater *ad libitum* food intake, compared with participants who consumed an alcohol-free placebo.

3.4.2. Method

3.4.2.1. Participants

Sample size was determined from previous investigations examining the effect of distraction on meal memory and subsequent food intake. Oldham-Cooper et al. (2011) found an effect size of d = 0.68 for the comparison between undistracted and distracted individuals on food intake and an effect size of d = 0.67 for meal memory between these two conditions. In order to detect an effect size of d = 0.67 with 80% power at an alpha level of 5%, 58 participants were required. Sixty participants were recruited to allow for any cases which may need to be excluded. Sixty participants (male = 30) aged between 18 and 62 y (M = 24.47, SD = 10.13) took part, and were recruited through online and email advertisement, and word-of-mouth. Participants were eligible to take part if they had no history of food allergies or intolerances, were not vegetarian or vegan, and were regular consumers of alcohol (consuming at least 10 UK alcohol units per week). Participants were excluded if they had a current or past alcohol use or eating disorder, had a current or recent illness that may increase sensitivity to alcohol (e.g., cold and flu), were taking medication that may be affected by alcohol, and were currently breastfeeding or pregnant. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences

Research Ethics Committee (reference number: 1737). Participants were reimbursed through either course credits or received a £10 shopping voucher.

3.4.2.2. Design

The study used a between-subjects, single-blind randomised design with drink type (alcoholic drink, placebo-alcohol) as an independent variable. The dependent variables were free recall and serial recall of the lunch meal, general memory recall, *ad libitum* intake (kcal) and total intake (test drink and *ad libitum* kcal combined).

3.4.2.3. Measures

Beverage Preparation and Administration. The present study used an alcohol dosage of 0.5 grams of alcohol per kilogram of participant bodyweight (g/kg) (4.47 UK units of alcohol for a participant weighing 70 kg). The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV) up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal) and was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. The placebo drink consisted of diet lemonade only; a vodka mist was sprayed on the surface of the drink to create the impression that it contained alcohol.

Lunch meal .The lunch meal used was similar to that used in a previous study (Oldham-Cooper et al., 2011). All lunch items were manufactured by Tesco's Ltd except for the potato chip snack (Hula Hoops; KP Snacks Ltd, Ashby-de-la-Zouch, United Kingdom). Nine foods were served one-by-one on separate plates in 90-second intervals, accompanied with a 250-gram serving of water. The foods were served in this way in order to match eating duration across foods and participants, and to measure how well participants remembered the order of the nine foods. Each plate was brought to the participants after they had finished the previous food. Participants were required to consume all of the lunch meal and could consume as much or as little of the water as they desired. See Table 3.1 for a list of the lunch items served.

Table 3.1. Lunch items served, in presentation order.

Food Item	Amount (grams)	Energy per portion (kcal)
Cheese twists	8	41
Ham sandwich[a]	34.96	94.10
Carrot batons	25	10.50
Mini Cornish pasty	30	104
Cheese sandwich[b]	34.96	125.20
Sausage Roll	11	34
8 Cherry tomatoes	70.71	14.14
Scotch egg	11.90	27.97
15 Potato chip snacks	12.71	64.08
Total	239.24	514.99

[a] Comprising half a slice of Tesco White Medium Bread (20 g), 5 g of Tesco Butterpak Spreadable Butter, 10 g of Tesco Everyday Value Cooked Ham.

[b] Comprising half a slice of Tesco White Medium Bread (20 g), 5 g of Tesco Butterpak Spreadable Butter, 10 g of Tesco Everyday Value Grated Cheddar.

Taste Test Preparation: The test meal consisted of a 200 g serving of Maryland chocolate chip cookies (487 kcal/100g) and a 250-gram serving of water. Cookies were broken into smaller pieces so that participant could not easily monitor the amount consumed (Higgs & Woodward, 2009). Taste-test consumption was calculated by subtracting the post taste-test weight from the pre-taste-test weight. Grams consumed was converted to kilocalories. The taste-test has been shown to be a valid measure of food intake (Robinson et al., 2017).

Free recall task: Participants were required to recall the nine food items they consumed during the lunch meal in no specific order. Using pen and paper, participants wrote down as many of the lunch items as they could remember. Two independent reviewers rated whether participants correctly recalled each of the nine lunch items, with an agreement of 94.45%. Disagreements in scoring was resolved by the lead author.

Serial order recall task: Participants were asked to recall the specific order in which the nine food items were presented.

Meal vividness rating: Participants were asked on a 100-mm Visual Analogue Scale (VAS) 'How vividly can you remember the lunch meal you ate earlier?' Anchored scores were 'Not At All' and 'Extremely'.

General Memory Measure: General memory performance was also measured. Participants were shown a wordlist consisting of 6 capital cities and 6 countries to memorise.

Dutch Eating Behavior Questionnaire: The Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien et al., 1986) is a 33-item questionnaire which measured eating styles associated with being overweight. The three subscales are restraint ($\omega t = .93$), emotional eating ($\omega t = .96$), and external eating ($\omega t = .90$).

Timeline Follow Back: In the Timeline Follow Back (TLFB; Sobell & Sobell, 1992), participants estimated the number of alcohol units consumed over the past 7 days, measuring typical drinking habits.

Alcohol Use Disorders Identification Test: The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is a 10-item questionnaire assessing hazardous drinking. Scores range between 0 and 40, with scores of ≥ 8 indicating hazardous alcohol use ($\omega t = .84$).

Snack Urge Scale: The Snack Urge Scale (SUS; Hardman et al., 2015) comprises four items which measured expected liking, desire to consume, craving, and difficulty to resist chocolate chip cookies. Each item was measured using a 100-mm VAS ('Not at all' – 'Extremely') and combined as a total snack urge score (maximum score of 400).

Appetite Ratings: (AR; Blundell et al., 2010) of hunger (I feel hungry) and fullness (My stomach feels full) were measured using a 100-mm VAS ('Not at all' – 'Extremely'). These scores were combined (hunger added to the inverse score of fullness) and reported as a single appetite rating (maximum score of 200).

Biphasic Alcohol Effects Scale: (BAES; Martin et al., 1993). The BAES is a 14-item scale which is comprised of two 7-seven item sub-scale, measuring the sedative and stimulating effects of alcohol.

Participants were required to rate the extent to which they are experiencing both sedative (e.g., down, inactive) and stimulatory feelings (e.g., elated, energized) at the present moment on a 10-point scale. Anchored scores are ‘Not at all’ and ‘Extremely’.

3.4.2.4. Procedure

Test sessions took place between 12:00 – 18:00 on weekdays in the Department of Psychology on the University of Liverpool campus. Sessions lasted approximately 120 minutes. The study was advertised as a study investigating ‘alcohol’s effect on memory and taste perception’. Participants were told that memory performance would be measured but were not told that memory of the lunch meal would be assessed. Prior to the beginning of the session, all participants were asked to consume a light meal not high in fat approximately an hour before the beginning of the test session. Upon arrival, participants were presented with the information sheet and provided informed consent. Participants were asked to report when they had last eaten and what they had consumed, before being breathalysed - all had a breath alcohol concentration (BrAC) of 0.00. Participants then completed a medical history questionnaire to assess whether they had any food allergies. Height and weight measurements were taken in order to calculate the alcohol dosage. Next, baseline appetite ratings and snack urge scale ratings were recorded, followed by completion of the DEBQ, AUDIT, TLFB and baseline BAES. Participants then consumed the test drink. They were required to consume the drink within 10 minutes, followed by a 10-minute absorption period where participants sat quietly. Next, a second breathalyser measure was taken, followed by a second set of BAES, appetite and snack urge ratings. Next, participants consumed their lunch meal. Afterwards, participants completed a third set of appetite, snack urge and BAES ratings. Participants were then presented with the word list for the general memory measure to memorise for one minute. This was measured in order to observe whether alcohol consumption successfully impaired general memory performance, as would be expected. Afterwards, participants took a 30-minute break where they were required to stay in the test room and to abstain from eating. Participants were offered light reading material during this time. After the break, participants were given one minute to recall items from the word list, before completing another breathalyser measure and appetite and snack urge ratings. Participants then completed the

taste test for 10 minutes. During this period, participants were asked to taste the test food as much or as little as they wanted, and to provide ratings based on certain characteristics of the foods (data on ratings were not analysed). Afterwards, BAES ratings were taken again. Participants were then given three minutes to complete the free recall lunch item task, followed by three minutes to complete the serial order recall task. The lunch memory measures were completed after the taste test to avoid cueing participants of their lunch meal. Participants then completed the vividness rating, and an awareness check. Finally, participants were fully debriefed and reimbursed for their time. See Table 3.2. for overview of the study procedure.

Table 3.2. Overview of the procedure. With approximate timings and durations of each task.

Task/Measure	Start Time (Minutes Post-arrival)	Duration (in minutes)
Information Sheet	0	1
Consent Form	1	2
Baseline breathalyser measure	3	1
Medical History Questionnaire	4	3
Height and Weight Measurement	7	2
Baseline Appetite Ratings	9	0.5
First Snack Urge Questionnaire	9.5	0.5
Dutch Eating Behaviour Questionnaire	10	3
Alcohol Use Disorders Identification Test	13	1
Timeline Follow-back Questionnaire	14	2
Baseline BAES	16	1
Consumption of Drink	17	10
Absorption Period	27	10
Second Breathalyser Measure	37	1
Lunch Meal	38	13.5
Post-lunch Appetite Ratings	51.5	0.5
Second Snack Urge Questionnaire	52	0.5
Second BAES	52.5	1
Memorise Word List	53.5	1
Break	54.5	30
Word List Recall	84.5	1
Third Breathalyser Measure	85.5	1
Third Hunger, Fullness, & Thirst Ratings	86.5	0.5
Third Snack Urge Questionnaire	87	1
Third BAES	88	1
Taste Test	89	10
Fourth BAES	99	1
Fourth Breathalyser Measure	100	1
Free Recall Task	101	3
Serial Recall Task	104	3
Vividness Rating	107	0.5
Awareness Check	107.5	2
Debrief Sheet	109.5	2
Reimbursement	111.5	2

3.4.2.5. Data Analysis

Analyses were performed using SPSS 25 (IBM Corporation, Armonk, NY, USA). We performed independent t-tests to test for any significant differences between conditions in the meal memory measures, general memory measure, food calorie intake and total calorie intake (cookie and drink calories combined). Mixed ANOVAs were conducted to observe differences between drink conditions and differences across time for appetite ratings, snack urge ratings and BAES stimulation and sedation ratings (see findings of snack urge ratings and BAES ratings in Appendix A). Sex differences were also investigated: 2 (sex; male, female) x 2 (drink; placebo, alcohol) between-subjects ANOVAs were conducted on cookie intake, general memory performance, and meal memory measures. A sensitivity analysis revealed that removing these participants from all analyses did not affect the statistical significance of the results. The method and analysis strategy for Study 1 was pre-registered on the Open Science Framework (see protocol here: osf.io/mbxs8/).

3.4.3. Results

3.4.3.1. Participant characteristics

Means and standard deviations are displayed in Table 3.3. Independent t-tests revealed no significant differences between groups on any of the measures included in Table 3.3.

Table 3.3. Sample characteristics and baseline scores, split by drink condition (mean ± SD)

	Alcoholic drink (N = 30)	Placebo-Alcohol (N = 30)
Age (y)	23 ± 9.72	25.93 ± 10.48
AUDIT (out of 40)	10.80 ± 5.03	10.97 ± 5.25
BMI (kg/m^2)	23.57 ± 3.84	25.81 ± 5.25
DEBQ Emotional	2.42 ± 0.93	2.52 ± 0.68
DEBQ External	3.38 ± 0.55	3.29 ± 0.65
DEBQ Restraint	2.38 ± 0.87	2.31 ± 0.68
7-day TLFB (alcohol units)	16.93 ± 11.61	16.20 ± 10.97
Baseline Appetite (out of 200)	84.07 ± 42.39	73.43 ± 37.10
Baseline Snack Urge (out of 400)	204.70 ± 82.48	177.17 ± 63.89
Baseline Sedation BAES (out of 49)	17.73 ± 10.97	16.17 ± 11.51
Baseline Stimulation BAES (out of 49)	33.37 ± 10.79	34.33 ± 8.41

AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index; DEBQ = Dutch Eating Behaviour Questionnaire; TLFB = Timeline Follow-back; BAES = Biphasic alcohol effects scale.

3.4.3.2. Calorie Measures (Figure 3.1)

There was no significant difference between drink conditions on the amount of calories consumed during the taste test $t(58) = 1.31$, $p = .196$, $d = 0.34$. However, participants in the alcohol drink condition consumed significantly more calories than the placebo-alcohol condition when combining those from both the drink and cookies consumed $t(58) = 5.55$, $p < .001$, $d = 1.43$. See Figure 3.1 for caloric intake split by drink condition. Furthermore, a 2 (sex; male, female) x 2 (drink; placebo-alcohol, alcohol) between-subjects ANOVA on cookie intake found a main effect of sex, whereby males consumed significantly more cookies than females $F(1, 56) = 13.25$, $p = .001$, $\eta_p^2 = .19$, but a nonsignificant main effect of drink $F(1, 56) = 1.41$, $p = .241$, $\eta_p^2 = .02$, and a nonsignificant sex by drink interaction $F(1, 56) = 0.04$, $p = .846$, $\eta_p^2 < .01$.

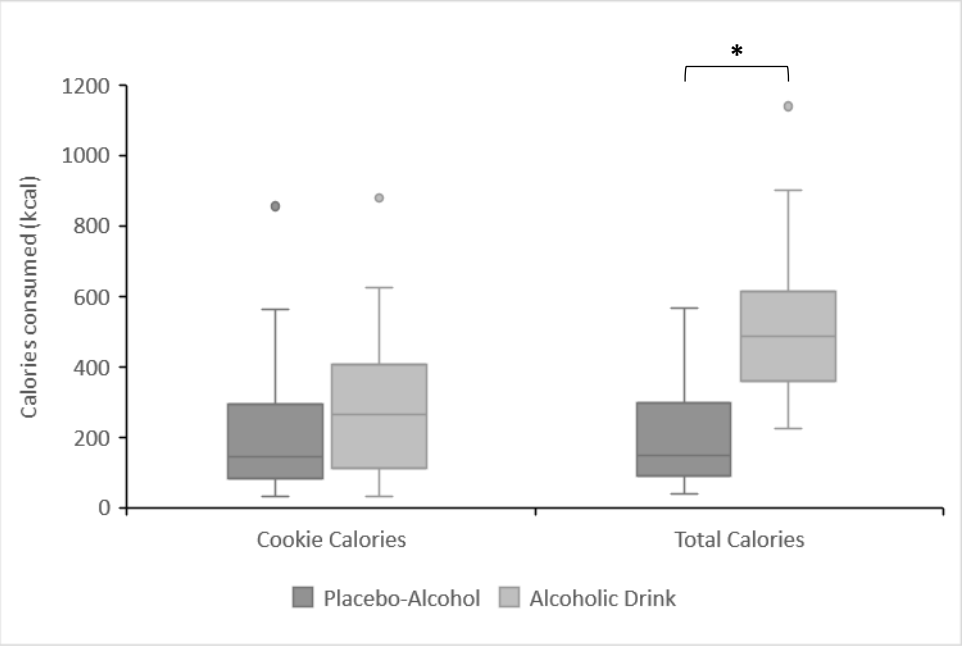

Figure 3.1. Boxplot displaying number of calories consumed during the *ad libitum* taste test (cookie calories) and combined with calories consumed from the test drink (total calories), split by condition. Dots indicate outliers.
Note. *$p < .001$

3.4.3.3. Memory measures (Table 3.4)

There was no significant difference between drink conditions for vividness ratings $t(58) = 0.34$, $p = .735$, $d = 0.09$, general memory recall $t(58) = 1.68$, $p = .098$, $d = 0.43$, for serial-order recall $t(58) = 0.92$, $p = .362$, $d = 0.24$, or for the free-recall lunch item task $t(58) = 1.66$, $p = .103$, $d = 0.43$. Similarly, performing separate 2 (sex; male, female) x 2 (drink; placebo-alcohol, alcohol) between-subjects ANOVAs revealed no significant main effects or interaction effects for any memory measure ($p > .05$). See Table 3.4 for scores on memory measures.

Table 3.4. Scores on outcome measures, split by drink condition (mean ± SD) *Note.* *p < *.05*

	Alcoholic Drink (N = 30)	Placebo-Alcohol (N = 30)
Vividness Rating (out of 100)	71.93 ± 14.80	73.13 ± 12.44
General Memory Recall (out of 12)	7.33 ± 2.07	8.20 ± 1.92
Lunch Item Recall (out of 9)	7.43 ± 1.57	8.07 ± 1.39
Serial Order Recall (out of 9)	4.60 ± 2.21	5.13 ± 2.29
Drink volume (mean ± SD; ml)	473.81 ± 88.81	510.76 ± 123.33
Drink volume (minimum and maximum; ml)	337.28 – 737.12	268.56 – 800
Drink calories (kcal)	229.31 ± 42.95	4.88 ± 1.18
Cookie intake (kcal)	284.96 ± 204.77	218.81 ± 186.03
Total intake (drink and cookies combined; kcal)	514.27 ± 217.75*	223.70 ± 186.38*

3.4.3.4. Appetite Ratings (Figure 3.2)

A 2 (drink; placebo-alcohol, alcoholic drink) x 4 (time; baseline, post-drink, post-lunch, pre-taste test) mixed ANOVA was conducted with drink as a between-subjects factor and time as a within-subjects factor. Mauchly's test indicated that the assumption of sphericity had been violated for the main effect of time $\chi^2(5) = 23.25$, $p < .001$, Greenhouse-Geisser corrected tests are reported ($\varepsilon = .828$). The analysis revealed a significant main effect of time $F(2.48, 141.62) = 78.83$, $p < .001$, $\eta_p^2 = .58$. Bonferroni pairwise comparisons revealed that baseline appetite ratings were significantly higher than post-lunch ($p < .001$; mean difference = 51.36; 95% CI [36.99, 65.71]) and pre-taste test ratings ($p < .001$; mean difference = 39.53; 95% CI [27.63, 51.33]). Post-drink ratings were also significantly higher than post-lunch ($p < .001$; mean difference = 63.29; 95% CI [48.64, 77.77]) and pre-taste test ratings ($p < .001$; mean difference = 51.46; 95% CI [36.54, 66.14]). Post-lunch ratings were shown to be significantly lower than pre-taste test ratings ($p = .006$; mean difference = 11.83;

95% CI [-21.15, -2.58]). The analysis also revealed a nonsignificant main effect of drink type $F(1, 57) = 2.67$, $p = .108$, $\eta_p^2 = .05$ and a nonsignificant drink type by time interaction $F(3, 171) = .78$, $p = .504$, $\eta_p^2 = .01$. See Figure 3.2 for appetite scores across time points, split by condition. See Appendix A for full list of means and standard deviations of appetite ratings at each time point, split by condition.

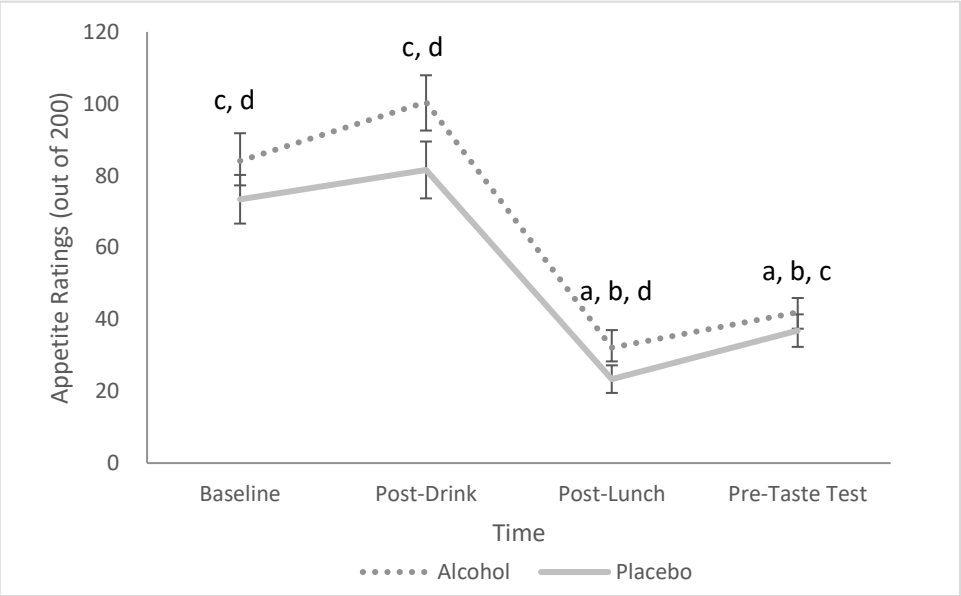

Figure 3.2. Appetite Ratings split by time and drink condition (Mean ± SEM). Letters refer to Bonferroni corrected pairwise comparisons breaking down significant differences ($p < .05$) between time points: a = difference from baseline; b = difference from post-drink; c = difference from post-lunch exposure, d = difference from pre-taste test.

3.5. Interim discussion

Study 1 found that consumption of an alcoholic drink did not significantly affect performance on the free-recall food memory task, serial-recall food memory task or ratings of meal vividness. Therefore, our prediction that alcohol consumption can impair meal memory is rejected. Findings also revealed that consumption of the alcoholic drink did not significantly decrease performance on the general memory task, nor did it significantly alter *ad libitum* consumption of cookies. This goes against our prediction that alcohol consumption would decrease general recall and increase food intake.

One explanation for failing to find a significant difference in all memory measures may have been due to the alcohol dosage used. Previous studies investigating the effect of alcohol intoxication on delayed recall typically use higher doses than the one used in the present experiment (Söderlund et al., 2005; Sutker et al., 1983). This is important because research has shown that memory impairment can occur in a dose-dependent manner (Bisby et al., 2010). Furthermore, by minimising alcohol expectancy effects between the two conditions by using an alcohol-free placebo, the difference in recall may have been smaller than in a more naturalistic context, where individuals are aware when a drink is alcoholic or not. However, previous research suggests alcohol expectancy has a small effect on information processing (Hull & Bond, 1986).

Findings also showed no significant association between performance on the free-recall meal memory task and subsequent food intake. Several studies have failed to identify the components of meal memory which directly affect subsequent food intake. It is plausible that the aspects of meal memory measured in Study 1 may not be relevant to subsequent food intake. Other studies have used measures which focus on recalling the quantity of a lunch meal (Mittal et al. 2011; Whitelock et al., 2018a; Whitelock et al., 2019) and recalling feelings relating to interoceptive states, such as hunger (Brunstrom et al., 2012; Whitelock et al., 2018a; Whitelock et al., 2019). These may be more important and relevant components of meal memory which help guide subsequent eating episodes, as compared with the current measures used.

Study 1 has a few limitations. Firstly, participants in the alcohol condition completed the recall measures when they were still intoxicated. It is therefore not possible to confirm whether impairments of memory performance were the result of disruption during the encoding or the retrieval phase, this limitation could be overcome by incorporating a longer delay between alcohol consumption and subsequent recall. Furthermore, Study 1 was not able to isolate the effect of impaired memory on subsequent food intake. Alcohol intoxication influences many factors which can increase food consumption, such as inhibitory control (Christiansen et al., 2016) and reward processing (Schrieks et al., 2015). As participants were still intoxicated during the taste test, the two

conditions were unmatched on a number of confounding factors. Given these issues, Study 2 looked to build upon the current findings and to address the mentioned limitations.

3.6. Study 2

3.6.1. Overview

Study 2 investigated whether other forms of meal memory may be disrupted by alcohol intoxication and alter later food intake. We chose to measure participants' visual memory of the portion size of a meal consumed, vividness of a meal and memory of satiety experienced after a meal. Furthermore, we used a greater alcohol dosage - 0.6 g/kg - and informed participants in the alcohol-free condition that they would be consuming a soft drink. This was done to produce a more naturalistic measure of alcohol's effect on delayed recall (combining both pharmacological and expectancy effects). We also incorporated a longer interval between consumption of the test drink and subsequent recall in order to allow for participants to have a lower alcohol level at the point of recall.

An additional aim was to investigate whether alcohol consumed *after* a lunch meal may in fact *enhance* meal memory. The ability for alcohol to influence episodic memories may depend on whether information is presented before or after consuming alcohol. As previously mentioned, research has shown that alcohol impairs learning when intoxication occurs at the encoding stage (when alcohol is consumed before information is presented). However, alcohol can *enhance* learning when intoxication occurs *after* the encoding stage and during consolidation (when alcohol is consumed after information is presented; Knowles & Duka, 2004; Parker et al., 1980; Weafer et al., 2016). For example, Weafer et al. (2016) found that alcohol consumed after presentation of stimuli significantly improved recall compared with consumption of a placebo-alcohol, suggesting that alcohol consumption can aid consolidation of recent memories and boost later recall. This phenomenon, termed 'retrograde facilitation' is believed to occur due to the ability of alcohol intoxication to protect memories formed prior to alcohol consumption by impairing the ability to form new memories, and therefore reduce interference once alcohol has been consumed (Wixted, 2005).

Alcohol consumed after a meal may therefore increase the quality of episodic memories relating to the meal, compared with when alcohol is consumed before the meal, and when alcohol is not consumed.

To investigate the effect of the timing of the alcoholic drink in relation to the meal, three conditions were implemented. Participants either 1) consumed an alcohol-free drink before consuming a lunch meal (soft drink condition), 2) consumed an alcoholic drink before consuming a lunch meal (pre-meal drink condition), or 3) consumed an alcoholic drink after consuming a lunch meal (post-meal drink condition). After a break (2 hours long in the post-meal drink condition, 2.5 hours long in the soft-drink and pre-meal drink condition), participants were given *ad libitum* access to chocolate chip cookies and meal memory recall was measured. We predicted that meal memory would be greatest in condition three and lowest in condition two, and therefore we also predicted that *ad libitum* food intake would be lowest in condition three and greatest in condition two. We also tested for general memory performance of words and predicted that recall of words presented before the test drink would be greater in the two alcohol conditions as compared with the soft drink condition. Conversely, we predicted that recall of words presented after the test drink would be poorer in the two alcohol conditions compared with the soft drink condition.

3.6.2. Method

3.6.2.1. Participants

Sample size was calculated based from previous research examining the enhanced effect on memory consolidation after alcohol consumption. A previous study found that alcohol consumption after viewing neutral stimuli during consolidation produced a large effect on memory recall (Weafer, Gallo & De Wit, 2016; $d = 0.79$). In order to detect a comparable effect with 80% power, $\alpha = 0.05$, 66 participants were required. We aimed to recruit 72 participants which would allow us to detect a large effect size ($d = 0.76$) at 80% power, $\alpha = 0.05$. To power for food intake, the design controlled for between-subject differences in food intake by incorporating a baseline session, whereby *ad libitum* food intake was measured and included as a covariate when comparing differences in food intake. This analysis strategy has been used in previous research (e.g., Gadah, Brunstrom, & Rogers, 2016). We used this analysis in order to reduce the between-subjects variance of food intake without

implementing a within-subjects design. With 72 participants, we were powered to detect an effect size of $d = 0.5$ for differences in food intake at 80% power, $\alpha = 0.05$. In total, 73 participants were recruited due to one participant failing to attend the second session. After excluding this participant, 72 (male = 36) participants aged between 18 and 60 y (M = 24.31, SD = 9.51) were included in all data analyses. Participants were recruited through online and email advertisement, and word-of-mouth. The inclusion criteria were the same as in Study 1, except participants were required to typically consume at least 15 UK alcohol units per week. This was increased due to the larger alcohol dosage implemented in Study 2. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee (reference number: 3116). Participants were reimbursed through either course credits or a £20 shopping voucher.

3.6.2.2. Design

The study used a between-subjects, single-blind randomised design with drink type (soft drink, pre-meal drink, and post-meal drink) as an independent variable. All participants attended two sessions. In the first (baseline) session, participants completed the same procedure and consumed a soft drink, followed by a lunch meal and then an *ad libitum* taste test. A week later, participants then completed the procedure in their randomly assigned condition. The dependent variables in session 2 were the number of calories consumed during the *ad libitum* taste test, total calories consumed (taste test calories and drink calories combined), meal vividness rating, memory for satiety, visual memory of the portion size of the lunch meal, and general memory recall.

3.6.2.3. Measures

Beverage Preparation and Administration. The present study used an alcohol dosage of 0.6 g/kg (5.37 UK units of alcohol for a participant weighing 70 kg). The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV) up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal). The drink was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. The soft drink consisted of diet lemonade only, and the volume was matched for body weight such that participants weighing the same would consume the same total volume of liquid in either

condition. All participants were told that they were consuming an alcohol-free diet lemonade drink during the first session, as were participants who were in the soft drink condition in session two.

Lunchtime meal.

Due to a manufacturing change in the caloric content of the lunch meal partway through the study, 13 participants consumed a lunch meal consisting of a 262.39 g serving of cheese and tomato pasta salad (Tesco UK). The remaining 59 participants consumed a 250.93 g serving of the same Tesco brand cheese and tomato pasta salad to ensure that all lunches were matched on caloric content (1.79 kcal per gram; 450 kcal per serving). The lunch meal was divided into six equicaloric portions, served one at a time in 90-second intervals to control for meal duration. Each plate was brought to the participant by the experimenter after they had finished eating the food of the previous plate. Participants were required to consume all of the lunch meal in both sessions. A 250 gram serving of water was provided with the lunch meal which participants could consume as much or as little of. The same lunch meal was served in both session 1 and 2.

*Taste Test Preparation***:** The same as in Study 1.

Meal vividness rating (Session 2): The same as in Study 1.

Picture presentations (Sessions 1 and 2): To bolster the cover story and to measure general memory performance, participants were required to provide visual ratings of different images in both sessions 1 and 2. Participants were exposed to one set of images in session 1, and two sets in session 2 (one before consumption of the test drink and one after). Pictorial stimuli were taken from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1997). Images across the three picture sets consisted of objects, animals and people. Each set consisted of 24 images, each presented with a text label below which provided a name of the image (e.g., an image of an astronaut would have the text label ‘Astronaut’ displayed below it). All three presentations were matched on valence and arousal ratings (scored out of 9): session 1 picture set: valence = 5.91; arousal = 3.95, session 2 picture set A: valence = 5.99; arousal = 3.71, session 2 picture set B: valence = 5.95; arousal = 3.89. The order of picture sets in session 2 were counterbalanced. For each set, images were presented

alone with the text label for 5 seconds. Afterwards, the image and text label were presented on the left hand-side of the screen, and three rating scales on the right-hand side, this stayed on screen for 15 seconds. Participants were asked to rate the content of the image on three scales – 'calm/excited', 'unpleasant/pleasant', 'not dominant/dominant' (data not analysed).

General memory recall (Sessions 1 and 2): A surprise free recall based on the picture presentation was implemented in both sessions. The surprise element ensured consistency between the general and meal memory recall tasks. Participants were given 5 minutes to recall as many of the picture text labels as they could remember from the session 1 picture set at the end of the first session, and from both the session 2 set A and B picture presentations at the end of the second session. Participants were told to recall the exact text of each label in any order they wished, and to avoid recalling any related words or synonyms. A response was marked as correct if it was the same text, with the exception of pluralising the word or recalling the text label correctly, but with incorrect spelling. The dependent variable was the number of text labels correctly recalled for each presentation set.

Dutch Eating Behavior Questionnaire (Session 1): The same as in Study 1. The three subscales are restraint (ωt = .96), emotional eating (ωt =.95), and external eating (ωt =.90).

Expected Satiety Memory measure (Session 2): To measure memory for satiety, participants completed a computerised task in which they were asked to select the portion size of 18 meal foods to indicate the amount of food that would be required to produce the sensation of fullness that they experienced after lunch; adapted from Brunstrom et al. (2008). Food pictures started at 20 kcal and increased in 20 kcal increments up 1000 kcal. Participants completed this measure twice in session 2: once immediately after consuming their lunch meal and again at the end of the test session. The outcome measure for this task was the absolute score of the average of the kcal differences of the portion sizes selected between the two measures. A score of zero means there was no difference in portion size selection between the two time points, indicating perfect memory, larger scores indicate poorer memory. Participants were also asked whether they had consumed each of the food items to check for familiarity (referred to as the familiarity task in the procedure section).

Visual memory for portion size (Session 2): Participants were presented with a large bowl of pasta salad (twice the amount of the same pasta salad they were served for lunch). Participants were asked to self-serve the amount of food which they believe they were served earlier for lunch, from the bowl onto a plate. The outcome measure was the difference between the amount of pasta self-served and the actual amount of pasta served at lunch, converted into an error percentage (a percentage of zero indicating zero difference). A larger error percentage indicates a greater difference between the amount of pasta self-served and the actual amount served for lunch, indicating poorer memory for portion size.

Timeline Follow Back (Session 1): The same as in Study 1.

Alcohol Use Disorders Identification Test (Session 1): The same as in Study 1. ($\omega t = .82$).

Snack Urge Scale (Session 2): The same as in Study 1

Appetite Ratings (Session 2): The same as in Study 1.

3.6.2.4. Procedure

Test sessions took place between 13:15 and 18:30 on weekdays in the Department of Psychology on the University of Liverpool campus. The study was advertised as investigating 'alcohol's effect on visual and taste perception'. Prior to both session 1 and 2, participants were told to consume a light meal not high in fat approximately an hour before the beginning of each session. Upon arrival of session 1, participants were presented with the information sheet and provided informed consent. Participants were then asked to report when they had last eaten and what they had consumed. Participants then completed a medical history questionnaire to assess whether they had any food allergies. Height and weight measurements were then taken in order to calculate the volume of drink to be consumed. Next, participants consumed the test drink (a soft drink for all participants) in three separate servings in 5-minute intervals. Afterwards, a 10-minute absorption period was completed whereby participants sat quietly. Next, participants consumed the test meal (participants were required to consume the entire meal in both sessions 1 and 2), and then completed the picture presentation task. Afterwards, participants completed the AUDIT and TLFB. Next, there was an

approximately 132-minute break during which participants were asked to abstain from eating. We incorporated a longer break in Study 2 in order to further reduce alcohol levels which may otherwise confound subsequent recall, and did so in session 1 for consistency across sessions. After the break, participants completed the taste test, general memory recall task and DEBQ.

After at least 1 week, participants completed session 2. Firstly, participants completed a baseline breathalyser measure (all had a BrAc of 0.00), and baseline appetite and snack urge ratings. For participants in the soft drink and pre-meal drink conditions, they were then shown the pre-drink picture presentation and consumed their test drink (served in the same way as in session 1), followed by a 10-minute absorption period. They were then shown the post-drink picture presentation. Afterwards, they consumed their lunch meal before completing the first expected satiety memory task, and a second set of appetite and snack urge ratings. Next, participants completed a 2.5-hour break where they were asked to stay in the building and to abstain from eating. Participants in the soft drink condition were given the option of staying in the building or leaving and coming back after the break due to there being no ethical requirement to stay.

For participants in the post-meal drink condition, after completing the baseline ratings, they were shown the pre-drink picture presentation, then consumed their lunch meal, followed by the first expected satiety memory task and ratings of appetite and snack urge. Next, they consumed their test drink, followed by an absorption period and were then shown the post-drink picture presentation, followed by a 2-hour break. The break duration was calculated such that the inter-meal interval between the lunch meal and taste test was the same across conditions (160 minutes). After the break, participants in all conditions completed a new set of appetite and snack urge ratings and then the taste test. This was followed by the general memory recall task, the second expected satiety memory task and its familiarity task, the visual memory for portion size task, vividness rating, awareness check, study debrief and reimbursement. See Figure 3.3. for a schematic overview of the procedure for sessions 1 and 2.

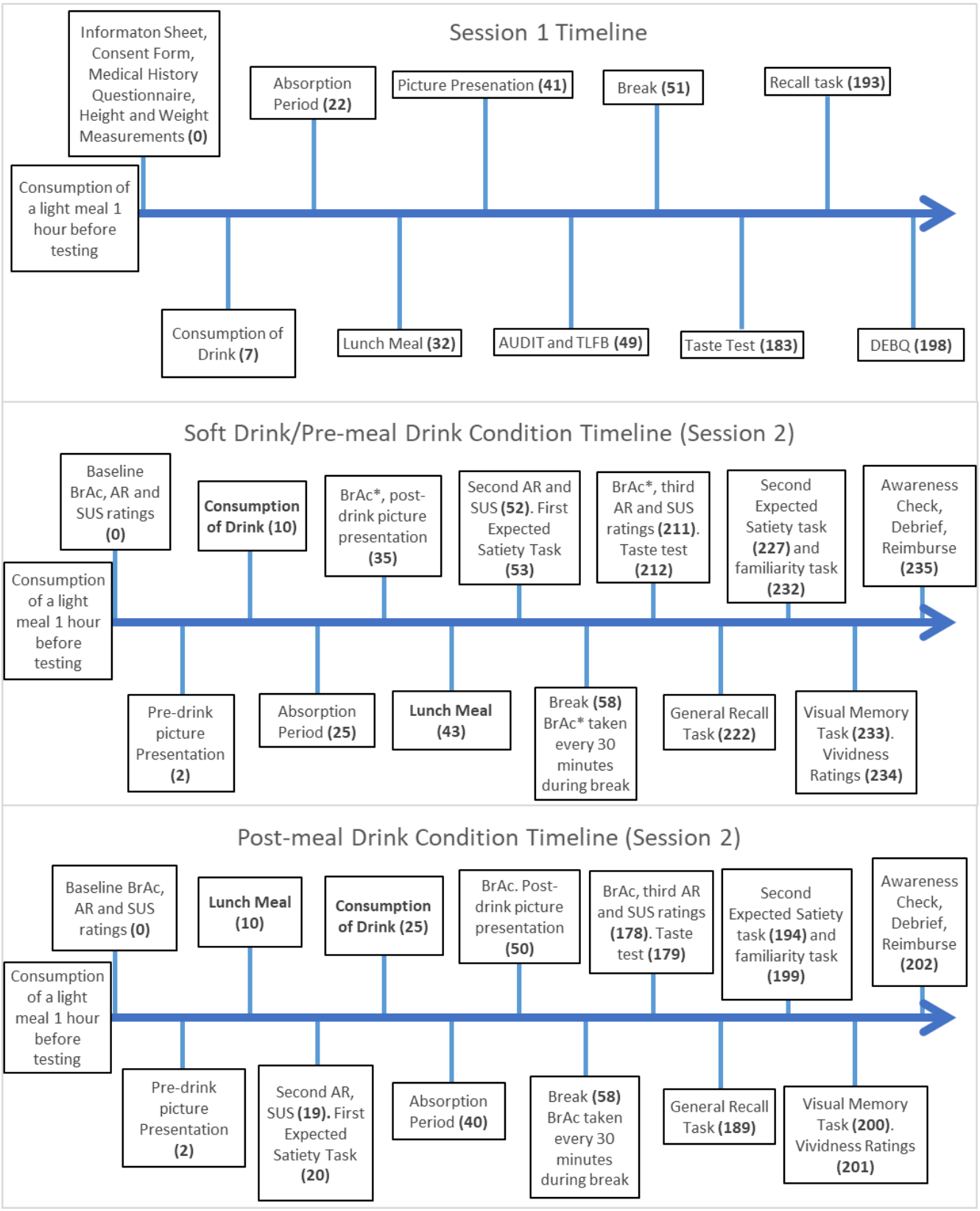

Figure 3.3. Schematic overview of the procedures for session 1 and session 2. Note. The procedure of session 1 was identical for all participants. Number in brackets represents the time (minutes) at which the task/measure was performed (relative to the start of the session). AR = Appetite Ratings; SUS = Snack Urge Scale ratings; BrAc = measure of breath alcohol concentration. *The procedure in the soft drink condition was identical to the pre-meal condition, except no BrAc measures were taken apart from at baseline. Timings are an approximation.

3.6.2.5. Data Analysis

We analysed food intake using an ANCOVA with drink as the between-subjects factor and baseline (session 1) caloric cookie intake as a covariate, the same ANCOVA was performed with sex included as a between subjects factor to investigate sex differences in food intake. Performance on each meal memory measure was compared across drink conditions using one-way ANOVAs, sex was included separately to investigate sex differences in meal memory performance. For the expected satiety memory measure, foods which had been previously consumed by less than 50% of participants were excluded from this analysis, as has been done in previous research (Whitelock et al., 2018a). Only 33.80% of participants had previously consumed grilled fish, therefore this item was excluded, leaving 17 food items for the analysis. For the general memory task, a mixed-design ANOVA was conducted to test for a drink by set interaction effect. Mixed ANOVAs were conducted to observe differences between drink conditions and differences across time for appetite ratings and snack urge ratings (see findings of snack urge ratings in Appendix A). Data for cookie intake from one participant from session 2 was lost due to human error, one participant did not complete the AUDIT questionnaire and one participant did not complete post-lunch snack urge ratings.

3.6.3. Results

3.6.3.1. Participant characteristics

Means and standard deviations are displayed in Table 3.5. Separate univariate ANOVAs found no significant differences between drink conditions in any of the sample characteristics included in Table 3.5.

Table 3.5. Sample characteristics split by drink condition and sex (mean ± SD).

	Soft Drink Male (N = 12)	Soft Drink Female (N = 12)	Soft Drink Total (N = 24)	Pre-meal Drink Male (N = 12)	Pre-meal Drink Female (N = 12)	Pre-meal Drink Total (N = 24)	Post-meal Drink Male (N = 12)	Post-meal Drink Female (N = 12)	Post-meal Drink Total (N = 24)
BMI (kg/m^2)	25.92 ± 3.80	24.47 ± 4.18	25.19 ± 3.97	23.65 ± 4.83	25.62 ± 4.95	24.64 ± 4.89	25.37 ± 4.34	22.38 ± 3.07	23.88 ± 3.98
Age (y)	31.83 ± 15.26	23.42 ± 9.57	27.63 ± 13.17	22.67 ± 5.30	24.33 ± 7.89	23.50 ± 6.63	23.17 ± 8.64	20.42 ± 3.26	21.79 ± 6.54
DEBQ Restraint	2.54 ± 1.18	2.85 ± 1.11	2.70 ± 1.13	2.17 ± 0.81	2.41 ± 0.77	2.29 ± 0.78	2.81 ± 1.08	2.33 ± 0.86	2.59 ± 0.97
DEBQ Emotional	2.29 ± 0.64	2.74 ± 0.97	2.52 ± 0.83	2.08 ± 0.54	2.42 ± 0.73	2.25 ± 0.65	1.84 ± 0.60	3.12 ± 0.80	2.46 ± 0.81
DEBQ External	3.11 ± 0.45	3.34 ± 0.52	3.23 ± 0.49	2.88 ± 0.60	3.30 ± 0.56	3.09 ± 0.61	3.02 ± 0.63	3.18 ± 0.77	3.07 ± 0.60
AUDIT (out of 40)	10.83 ± 8.39	9.92 ± 4.34	10.38 ± 6.55	10.42 ± 3.80	9.33 ± 4.03	9.88 ± 3.87	11.91 ± 5.82[1]	11.42 ± 4.42	11.65 ± 5.02[1]
7-day TLFB (alcohol units)	16.67 ± 15.89	15.00 ± 7.60	15.83 ± 12.21	20.50 ± 10.90	17.08 ± 6.55	18.79 ± 8.96	19.83 ± 9.04	16.75 ± 6.89	18.29 ± 8.02
Baseline General Memory Recall (Session 1; out of 24)	8.17 ± 2.21	9.58 ± 2.54	8.88 ± 2.44	8.92 ± 3.00	9.17 ± 3.56	9.04 ± 3.22	9.67 ± 2.23	9.75 ± 2.09	9.71 ± 2.12
Baseline Appetite (out of 200; Session 2)	112.17 ± 38.03	110.83 ± 39.67	111.50 ± 38.01	128.33 ± 24.34	125.67 ± 45.85	127.00 ± 35.93	103.08 ± 40.50	108.75 ± 30.71	105.92 ± 35.27
Baseline Snack Urge (out of 400; Session 2)	185.83 ± 63.89	204.42 ± 71.69	195.13 ± 67.09	192.08 ± 47.07	211.08 ± 82.24	201.58 ± 66.25	196.83 ± 44.90	234.58 ± 58.49	215.71 ± 54.52

Note. [1] = data missing from one participant. AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index; DEBQ = Dutch Eating Behaviour Questionnaire; TLFB = Timeline Follow-back

3.6.3.2. Calorie intake (Table 3.6):

An ANCOVA with baseline cookie intake as a co-variate revealed a non-significant main effect of drink on cookie intake $F(2, 67) = 0.49$, $p = .617$, $\eta_p^2 = .01$. Using the same ANCOVA model, total calorie intake significantly differed between drink conditions $F(2, 67) = 29.86$, $p < .001$, $\eta_p^2 = .47$. Bonferroni corrected pairwise comparisons revealed that total caloric consumption was significantly lower in the soft drink condition compared with both the pre-meal drink ($p < .001$; mean difference = 324.83 kcal; 95% CI [-441.69, -207.97]) and post-meal drink condition ($p < .001$; mean difference = 313.89 kcal; 95% CI [-443.05, -195.72]). Total calorie intake did not differ between the pre-meal and post-meal condition ($p = 1.00$; mean difference = 10.95 kcal; 95% CI [-107.27, 129.16]). See Table 3.6 for caloric intake split by drink condition. The same ANCOVA with sex as a between subjects factor revealed a main effect of sex on food intake $F(1, 64) = 7.82$, $p = .007$, $\eta_p^2 = .11$, with males consuming more than females. There was a nonsignificant sex by gender interaction $F(2, 64) = 0.75$, $p = .477$, $\eta_p^2 = .02$.

3.6.3.3. Meal Memory measures (Table 3.6)

There was a significant main effect of drink on expected satiety memory scores $F(2, 69) = 4.67$, $p = .013$, $\eta_p^2 = .12$. Bonferroni corrected pairwise comparisons revealed that the error score (higher scores indicating poorer memory) was significantly greater in the pre-meal drink condition, compared with the soft drink condition ($p = .016$; 95% CI [-81.52, -6.42]) which was in line with our prediction. However, no other significant main effects of drink condition were found for any other meal memory measure. See Table 3.6 for performance on meal memory measures, split by drink condition. When examining sex differences using 2 (sex; male, female) x 3 (drink; soft drink, pre-meal drink, post-meal drink) between-subjects ANOVAs, the visual memory measure showed a significant main effect of sex $F(1, 66) = 5.43$, $p = .023$, $\eta_p^2 = .08$ with males displaying a greater error percentage than females, and a significant sex by drink interaction $F(2, 66) = 4.08$, $p = .021$, $\eta_p^2 = .11$. This interaction was due to men having a significantly greater error percentage in the soft drink condition $t(22) = 3.23$, $p = .004$, $d = 1.32$, but not in the other two conditions ($p > .05$). There were no

other significant main effects of sex or sex by condition interaction effects for any other meal memory measure.

Table 3.6. Outcome measures, split by drink condition (mean ± SD)

	Soft Drink (N = 24)	Pre-meal Drink (N = 24)	Post-meal Drink (N = 24)
Vividness ratings (Session 2; Out of 100)	80.25 ± 14.44	71.75 ± 18.62	79.13 ± 11.88
Expected satiety error (kcal)	63.68 ± 25.85[a]	107.65 ± 75.51[a]	71.91 ± 45.40
Visual Memory (%)	21.13 ± 14.28	14.75 ± 10.43	14.31 ± 10.26
Baseline *ad libitum* food Intake (kcal; Session 1)	292.94 ± 164.15	297.92 ± 135.33	280.72 ± 210.73
Drink volume (mean ± SD; ml)	615.72 ± 116.96	603.69 ± 124.04	583.36 ± 113.09
Drink volume (minimum and maximum; ml)	373.73 – 800	404.74 – 800	353.33 – 800
Drink calories (kcal)	5.86 ± 1.11	292.10 ± 60.02	282.28 ± 54.72
Ad libitum food Intake (kcal; Session 2)	358.19 ± 214.57	400.92 ± 196.84	383.52 ± 216.36[1]
Drink and *ad libitum* intake combined (kcal; Session 2)	364.05 ± 215.08[d,e]	693.02 ± 211.63[d]	667.25 ± 228.18[e,1]

Note. Means with the same letter indicate a significant difference between each other; $p < .05$, Bonferroni adjustment for multiple comparisons. [1] = data missing from one participant.

3.6.3.4. General memory recall (Figure 3.4):

For this analysis, we wanted to explore whether recall in the pre-drink set was greater in the two alcohol conditions relative to the soft drink condition, but greater in the soft drink condition relative to the two alcohol conditions in the post-drink set. Therefore, only the interaction effect is relevant. A 2 (set; pre-drink, post-drink) x 3 (drink; soft drink, pre-meal drink, post-meal drink) mixed ANOVA revealed a significant set by drink interaction $F(2, 69) = 8.26$, $p = .001$, $\eta_p^2 = .19$. Univariate ANOVAs were conducted for each set separately (see Figure 3.4 for general memory recall of the pre-drink and post-drink sets). A significant main effect of drink in the pre-drink set $F(2, 69) = 4.39$, $p = .016$, $\eta_p^2 = .11$ was found, whereby recall in the post-meal drink condition was significantly greater than in the pre-meal drink condition ($p = .029$; mean difference = 2.71; 95% CI [0.21, 5.21]). This was unexpected, as due to a predicted effect of retrograde facilitation, we expected recall in the pre-drink picture set to be significantly greater in both alcohol conditions (i.e. the pre-meal and post-meal conditions), compared with the soft drink condition. There was also a significant main effect of drink condition in the post-drink set $F(2, 69) = 11.03$, $p < .001$, $\eta_p^2 = .24$, whereby recall in the pre-meal drink condition was significantly lower than in both the soft drink condition ($p < .001$; mean difference = 3.46; 95% CI [1.65, 5.27]) and the post-meal condition ($p = .046$; mean difference =

1.83; 95% CI [0.03, 3.64]). There was a nonsignificant difference between the soft drink and post-meal conditions (p = .092; mean difference = 1.63; 95% CI [-0.18, 3.43]). See Figure 3.4 for scores on the general memory task, split by drink condition and set.

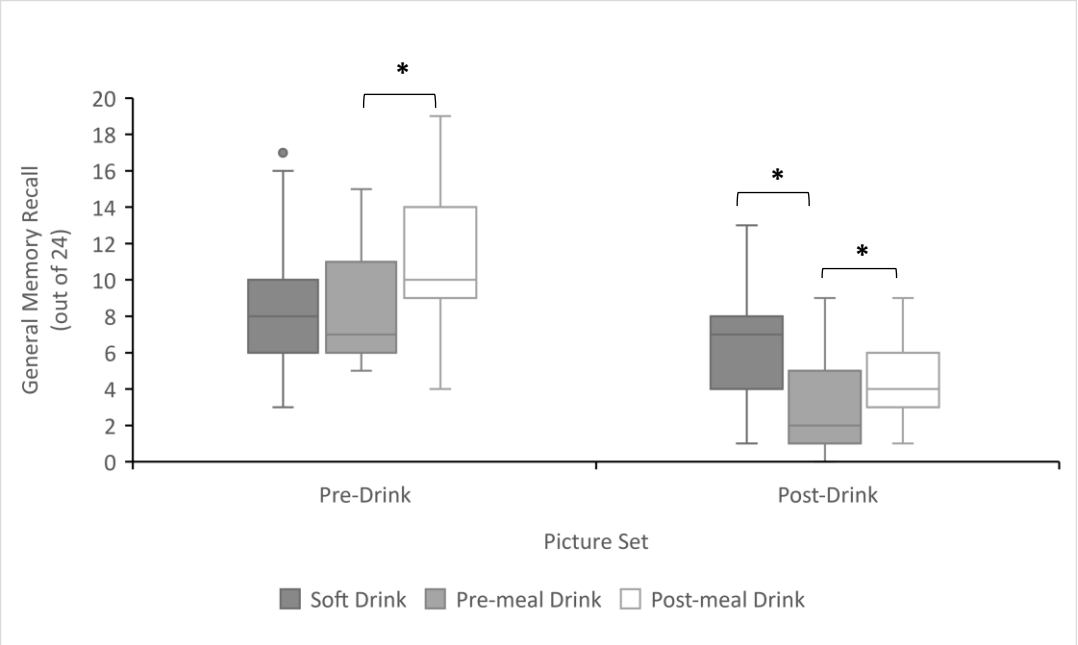

Figure 3.4. Boxplot displaying general memory recall split by the three drink conditions, and two set types in session 2. Dots indicate outliers. Note. *p < .05

3.6.3.5. Appetite Ratings (Figure 3.5)

A 3 (drink; soft drink, pre-meal drink, post-meal drink) x 3 (time; baseline, post-lunch, post-break) mixed ANOVA was conducted with drink as a between-subjects factor and time as a within-subjects factor. This revealed a main effect of time $F(2, 138) = 71.31$, $p < .001$, $\eta_p^2 = .51$. See Figure 3.5 for comparisons across time points. The analysis also revealed a significant main effect of drink $F(2, 69) = 4.01$, $p = .023$, $\eta_p^2 = .10$. Bonferroni corrected comparisons revealed that those in the soft drink condition had lower overall appetite ratings compared with the pre-meal drink condition (p = .029; mean difference = 22.31; 95% CI [-42.89, -1.72]) but did not significantly differ from the post-meal drink condition (p = 1.00; mean difference = 4.08; 95% CI [-24.67, 16.50]). Overall appetite ratings between the pre-meal and post-meal drink condition did not significantly differ (p = .100;

mean difference = 18.22; 95% CI [-38.81, 2.37]). Lastly, there was a nonsignificant drink by time interaction effect $F(4, 138) = 2.07$, $p = .088$, $\eta_p^2 = .06$. See Appendix A for a full list of means and standard deviations of appetite ratings at each time point, split by condition.

Figure 3.5. Appetite ratings split by condition and across each time point. (Mean ± SEM)
Note: Letters refer to Bonferroni corrected pairwise comparisons which compare appetite scores across each time point ($p < .05$): a = different from baseline scores; b = different from post-lunch scores; c = different from post-break scores.

3.7. General Discussion

Consistent with Study 1, Study 2 found that consumption of an alcoholic drink prior to consuming a lunch meal impaired meal memory when compared with consumption of a soft drink. In Study 2, this was evident for the measure of memory of satiety - participants in the pre-meal drink condition less accurately remembered the level of fullness experienced immediately after the lunch meal compared with those in the soft drink condition. However, an impairment was not evident for meal vividness ratings or visual memory of the portion size. Furthermore, the findings failed to show an enhanced recall of meal memory when the alcoholic drink was consumed after the lunch meal. There were also no significant differences in *ad libitum* food intake between the three conditions.

Therefore, our hypothesis that meal memory would be lowest in the pre-meal drink condition is only partially supported, with no support to show that this increased food intake. Furthermore, our hypothesis predicting that those in the post-meal drink condition would show the greatest meal memory and lowest food intake is rejected.

Study 2, but not Study 1 showed evidence that consumption of an alcoholic drink before a lunch meal can impair certain forms of meal memory compared with memory performance after consumption of an alcohol-free drink. Altering episodic memories of a recent meal is therefore an additional factor which is both caused by acute alcohol consumption and which, in other studies, has been shown to increase food intake. However, in both Study 1 and 2 we found no significant difference in food intake between drink conditions, therefore this proposition remains unsupported.

Findings also revealed that alcohol-induced changes to food intake and meal memory did not differ between men and women. This is in line with previous research which has shown that alcohol-induced food intake occurs in both males and females (Kwok et al., 2019). However, other research has shown alcohol-induced changes in immediate and delayed recall to be more impaired in women than in men (Jones & Jones, 1976; Jones & Jones, 2014; Niaura et al., 1987), therefore the present findings are not in line with previous studies showing a sex difference in memory impairments.

The present findings add to the literature by implementing a novel form of meal memory disruption. By using alcohol intoxication as a tool to manipulate and disrupt the encoding phase of memory formation, findings revealed that this was successful in altering the quality of some meal memories. It also provides support for previous literature which has shown that different methods of disruptions to memory encoding impair meal recall (Higgs & Woodward, 2009; Mittal et al., 2011; Oldham-Cooper et al., 2011). The present findings also highlight the difficultly in identifying the components of meal memory which are important in determining later food intake, as although a meal memory impairment was observed in both studies, food intake did not differ between conditions. However, this does not mean that meal memory is unimportant in determining food intake. Instead, it is possible that other components of meal memory, such as visual memory of portion size, may be a more important determinant in food intake. The memory manipulation used in the present study did

not appear to be strong enough in order to impair recall of all measured forms of meal memory, which may explain a lack of effect on food intake. Future research should continue to investigate which components of meal memory directly relate to subsequent food intake.

As discussed elsewhere (Whitelock et al., 2019), it is important to consider how motivated participants were to use recent memories of their lunch when deciding how much to eat in the taste test. One reason why unimpaired meal memory did not lead to a reduction in food intake could be due to the calorie content of the pre-load lunch meal. Pre-load meals in both Study 1 and 2 did not exceed 515 kcal. For some participants this may be considered a relatively small amount of food and therefore, after an inter-meal interval of 160 minutes (as was the case in Study 2), participants may not have felt motivated to restrict their food intake even when details of this lunch meal were well-remembered. There is some evidence to suggest gender differences may exist with regard to the effectiveness of manipulating meal memory on subsequent food intake. For example, the effect of focused attention has been established in female samples (Higgs & Donohoe, 2011; Robinson et al., 2014b), but is inconsistent in mixed gender samples (Seguias & Tapper, 2018; Whitelock et al., 2018a) and has not been shown in a male sample (Whitelock et al., 2019). One explanation for why Seguias and Tapper (2018) found a difference in food intake in a mixed-gender sample may be due to the caloric quantity of the pre-load used. In their study, participants were given *ad libitum* access to their lunch meal. This would have allowed participants to consume a personally 'normal' amount of food. This in turn may have resulted in the sample being more motivated to use episodic meal memories when deciding how much to consume at a subsequent eating episode. This suggestion is speculative, however future studies may wish to investigate how altering the personal appropriateness of a pre-load meal in terms of its caloric content, can moderate the effect of episodic memories on later food intake.

Findings of Study 2 failed to show evidence of enhanced meal memory recall when the meal was consumed prior to alcohol consumption. The magnitude of the retrograde facilitation effect may differ depending on the type of stimuli exposed to. For example, Weafer et al. (2016) found that the effect of consolidation was greatest for neutral stimuli (d = 0.79) compared with negative (d = 0.26)

and positive (d = 0.31) stimuli. It is plausible to assume that food-related stimuli may not be considered neutral. Therefore, as Study 2 was powered to detect a large effect size, we may have been underpowered to detect consolidation effects of other, non-neutral stimuli. However, we also did not find a consolidation effect for general memory recall, suggesting an overall failure in producing this effect.

Alternatively, a failure to detect enhanced meal memory may have resulted from the experimental design. By using the same test lunch in both the first and second session, participants may have established a clear memory of the lunch meal from the first session, allowing participants to remember back to the previous session to recall details of their lunch meal, therefore minimising the importance of the effect of the drink on memory formation. Although we incorporated a 1-week washout period to counter this issue, some participants may still have remembered the quantity of the lunch meal. This may explain why no differences were found for the visual memory and vividness measures. However, expected satiety memory was shown to be significantly impaired in the pre-meal drink condition relative to the soft drink condition. It may be that memory for fullness is more difficult to remember between sessions, compared with other forms of meal memory.

There are some limitations with Study 2. Firstly, during the break in the second session, participants in the soft drink condition were not required to wait in a waiting room during the break. Although all participants were told to abstain from eating, some participants in this condition would have had a different experience during their break compared to participants in the other conditions, although no significant difference in food intake was found. A second limitation was that during the recall phase, participants in both alcohol conditions were on the descending limb of the blood alcohol curve (see Appendix A for BrAc scores). The descending limb can produce sedation, negative mood (Babor et al., 1983; Lukas et al., 1986; Sukter et al., 1983) and impairment of certain forms of executive functioning (Pihl et al., 2003). One way to overcome this issue and to ensure participants were sober at the point of recall would have been to implement a longer delay of 24 or 48 hours after the exposure phase, which has been done in previous studies (Gawrylowicz et al., 2017; Weafer et al., 2016). However, we decided to implement a shorter period as this was essential in order to observe

the effect of meal memory on food intake. This is because previous research has shown that cueing participants of their lunch consumed on the previous day does not affect food intake, but cueing lunch which has been consumed on the same day reduces subsequent food intake (Higgs, 2002). This suggests that memories relating to food consumed only very recently can alter food intake. Therefore, a greater delay may have failed to tap into the effect of meal memory on food intake. Despite this, a difference in mood and executive performance may have contributed to a lack of enhanced recall through retrograde facilitation, which may have been observed otherwise with a longer delay.

In conclusion, both studies revealed that consuming a lunch meal whilst intoxicated can impair subsequent recall of certain lunch details. However, neither study provided evidence that meal memory predicted subsequent food intake. It therefore remains unclear as to whether alcohol induced changes to meal memory contribute towards alcohol-induced overeating.

Chapter 4: Investigating the interactive effect of food reward and impulsivity on alcohol-induced food intake

4.1. Overview

After conducting two studies which revealed that acute alcohol consumption can impair meal memory recall, but not affect food intake, Chapter 3 moved on to assess whether acute alcohol consumption increases food intake through the enhancement of food reward. Current evidence is inconsistent and suffers from methodological limitations. Therefore, the current chapter reports the findings of two studies which investigated whether food reward and food intake increase after acute alcohol consumption, and whether this effect may be dependent on the dose of alcohol consumed. Importantly, this was done such that a sufficient sample size was utilised, and homogenous methodology was implemented to allow for clearer comparisons between the two studies.

The study reported in this chapter is currently under review as: Gough, T., Christiansen, P., Rose, A., & Hardman, C.A (under review). The dose-dependent effect of alcohol on food-related attentional bias, food reward and intake.

4.2. Abstract

Acute alcohol consumption has been shown to increase food intake, and long-term alcohol consumption may be a risk for weight gain. A potential, but under-studied, mechanism for this effect is alcohol's ability to enhance food reward. In two studies, participants consumed an alcoholic drink (Study 3: 0.3 grams of alcohol per kilogram of bodyweight (g/kg); Study 4: 0.6 g/kg) and a placebo-alcohol drink in a within-subjects design. In both studies, food-related appetitive and motivational states, and attentional bias (AB) towards food-related cues were measured. In Study 3 (N = 44), participants completed a visual probe task with concurrent recording of eye-movements which measured AB towards images of palatable foods, unpalatable foods, and non-food control items. Participants also completed measures of appetite and snack urge ratings, salivary response towards palatable foods and an *ad libitum* food taste test. In Study 2 (N = 84), participants completed a similar procedure, but completed a modified Stroop task which measured differences in food-related and

alcohol-related AB across the two drink conditions. In Study 3, there was no difference in food-related AB between drink conditions, and no differences in snack urge ratings, appetite ratings, salivary response, or food intake. In contrast, Study 4 showed an alcohol-induced increase in AB towards food, but not alcohol. Snack urge, alcohol urge ratings and *ad libitum* food intake were also higher after alcohol consumption, relative to the placebo. Collectively, these findings suggest that alcohol can increase food reward and food intake, but these effects appear to be dose dependent.

4.3. Introduction

Obesity and over-consumption of alcohol are two major global health concerns, which may also be related, as excessive drinking has been implicated as having a causal role in the etiology of over-eating and obesity (Chapman et al., 2012; Sayon-Orea et al., 2011). This link between alcohol consumption and obesity is unsurprising given the high caloric density of alcohol at 7.1 kcal/g. Experimental evidence shows that not only are these calories poorly compensated for, but acute alcohol consumption can increase food intake relative to consumption of an alcohol-free drink (Kwok et al., 2019). One proposed mechanism for this alcohol-induced increase in food intake is the ability of alcohol to enhance the rewarding properties of food (Yeomans, 2010a).

In humans, food reward (defined as the momentary value of food; Rogers & Hardman, 2015) can be measured using explicit measures, such as self-report scales which measure appetite, liking of food and desire to consume food (Rogers & Hardman, 2015; Ruddock et al., 2017), but can also be measured using tasks which capture implicit biases to food cues such as measures of attentional bias. In the case of self-report measures, indices of food reward (i.e., appetite and snack urge ratings) have been shown to increase after alcohol consumption (Caton et al., 2004; Rose et al., 2015; Schrieks et al., 2015).

Attentional bias (defined as the ability for certain stimuli to capture one's attention; Field et al., 2016) has been implicated as an index of food reward because attentional biases are thought to indicate underlying appetitive motivational processes - when an object (such as food) is craved or

desired, a greater level of attention is allocated towards cues related to this object (for review, see Field et al., 2016). In support of this theory, several studies have demonstrated that attentional bias (AB) towards food cues is positively associated with motivational states relating to food, such as hunger and food craving (Castellanos et al., 2009; Gearhardt et al., 2012; Graham et al., 2011; Mogg et al., 1998; Nijs et al., 2010; Schmitz et al., 2014; Tapper et al., 2010; Werthmann et al., 2013; Werthmann et al., 2011). Furthermore, a recent meta-analysis by Hardman et al. (2020) found a significant correlation of $r = 0.13$ between food craving and food-related AB.

To date, little research has focused on how alcohol intoxication can alter food-related AB. One study found that AB towards food cues was increased by smelling alcohol odours, in the absence of alcohol consumption (Karyadi & Cyders, 2019). However, another study showed that the magnitude of food-related AB did not differ between consumption of a placebo-alcohol, and alcoholic doses of 0.3 g/kg or 0.65 g/kg (Monem & Fillmore, 2019). However, this study was powered to detect only a medium-to-large effect size, which may explain why no difference was found, as evidence suggests that the relationship between food craving and food-related AB is small (Hardman et al., 2020). Given these discrepant findings, the present research aimed to further investigate whether acute alcohol consumption can increase AB towards food cues.

The extent to which alcohol increases food-related AB may also depend on how rewarding the food cues are. Energy-dense, highly palatable foods (often high in fat and sugar) are more rewarding than low-calorie foods (Rogers & Brunstrom, 2016). Initial evidence suggests that alcohol can increase the desire to consume foods with low levels of palatability (Schrieks et al., 2015). However, there have been no studies to date which have systematically compared the effects of alcohol intoxication on AB towards high- and low-palatable foods. Alcohol intoxication may also produce changes in physiological responses to palatable foods. This is because cephalic phase responses (such as salivary response to food) have been shown to correlate with hunger (Wooley & Wooley, 1981) and desire to consume food (Keesman et al., 2016). Through alcohols' enhancement of food reward, salivary response to food cues may therefore increase after acute alcohol consumption, however this remains untested.

It has been suggested that acute alcohol consumption produces greater levels of food intake among individuals high in dietary restraint – those who restrict energy intake to avoid weight gain. This may occur due to a reduction in the ability to maintain restrained eating behaviours, resulting in a temporary change to dietary intentions (Caton, Nolan, & Hetherington, 2015). This effect was first studied by Polivy and Herman (1976a; 1976b) who found that when restrained eaters were aware of the presence of alcohol, their eating behaviour became disinhibited. Whereas, when restrained eaters were unaware of the presence of alcohol, food intake was suppressed (relative to unrestrained individuals), suggesting that alcohol-related expectancy effects may contribute towards disinhibited eating in restrained individuals. However, subsequent research has been unable to demonstrate that restrained eaters are more susceptible to alcohol-induced increases in food intake (Christiansen et al., 2016; Poppitt et al., 1996; Yeomans, 2010b; Yeomans, Hails, & Nesic, 1999), even when they are made aware of the presence of alcohol (Ouwens et al., 2003). Taken together, restraint is an important variable to take into consideration when conducting research on alcohol and food intake.

4.4. Study 3

4.4.1. Overview

Study 3 investigated whether food reward (measured using self-report appetite, snack urge ratings, salivary response to food, and AB towards food-cues) and *ad libitum* food intake would differ between administration of a placebo-alcohol and an alcoholic drink (dose = 0.3 g/kg). This dose was chosen because although Monem and Fillmore (2019) were unable to show an enhanced food AB at 0.3 g/kg, this same dose has been shown to enhance AB towards other appetitive stimuli (i.e., alcohol) relative to a placebo-alcohol (Duka & Townshend, 2004; Schoenmakers et al., 2008).

The AB measure was a visual probe task with concurrent eye-tracking, with comparisons of three image pairs: palatable food and unpalatable food images, palatable food and non-food images, unpalatable food and non-food images. Fixation duration from concurrent eye-tracking was the outcome measure as this has greater internal reliability as compared with reaction time assessments when measuring food-related AB using the visual probe task (van Ens et al., 2019). It was predicted that all measures of food reward and food intake would increase after consumption of an alcoholic

drink, relative to a placebo-alcohol. In secondary analyses, we tested whether dietary restraint moderates the effect of drink condition on food intake.

4.4.2. Method

4.4.2.1. Participants

At the time of data collection, no studies had investigated the difference in food-related AB between consumption of an alcoholic drink and placebo, therefore the study was powered to detect a small-to-medium effect size (d = 0.39) for differences in food-related AB between drink conditions. Using G*Power 3.1 (Faul et al., 2009) and based on 80% power and an alpha level of 5%, 43 participants were required. Forty-four participants (male = 22) aged between 18 and 54 y (M = 25.55, SD = 8.22), were recruited in order to achieve full counterbalancing of drink order. Participants were recruited through online and email advertisement, and word-of-mouth and were eligible to take part if they were aged 18 – 65 y, had no history of food allergies or intolerances, were regular consumers of alcohol (drinking at least 10 UK alcohol units per week), and enjoyed consuming cookies and tortilla chips, as these were used as test foods. Participants were excluded if: they wore glasses to correct their vision (due to interference with the eye-tracking camera); had a current or past alcohol use or eating disorder; had a current or recent illness that may increase sensitivity to alcohol (e.g., cold and flu); were taking medication that may be affected by alcohol; were currently breastfeeding or pregnant. Participants were also required to consume a light meal, low in fat, one hour prior to the test session. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee (reference number: 838). Participants were reimbursed through either course credits or a £10 shopping voucher.

4.4.2.2. Design

The study used a single-blind randomised within-subjects design with drink type (alcoholic drink, placebo-alcohol) as the independent variable. Each participant completed both conditions in two sessions, separated by at least one week. The order of conditions was randomised and counterbalanced across participants.

4.4.2.3. Measures

Beverage Preparation and Administration: The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV) at a dose of 0.3 g of alcohol per kg of body weight (2.68 UK units of alcohol for a participant weighing 70 kg), up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal). The drink was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. The placebo drink consisted of diet lemonade only (beverage volume was matched within participants across conditions); a vodka mist was sprayed on the surface of the drink to create the impression that it contained alcohol.

Pictorial Stimuli: The Visual Probe Task (VPT) consisted of three image types (with two subtypes within each image type, presented on an equal amount of trials) – palatable foods (tortilla chips and chocolate chip cookies), unpalatable foods (boiled potatoes and wholemeal bread), and non-food controls (leaves and drink coasters). This generated three types of image pairs – palatable and unpalatable, palatable and control, unpalatable and control (each with eight image pairs). To ensure that images were well matched on visual characteristics, tortilla chips, boiled potatoes and leaves were only ever presented with each other, and chocolate chip cookies, wholemeal bread and drink coaster were presented with each other. Images were sourced from a web browser (https://www.google.com/imghp?hl=EN) and selected if they had appropriate visual characteristics. All images were 400 x 300 pixels and were displayed on a plain black background.

Visual Probe Task (VPT): The VPT was programmed in Inquisit version 4 (Millisecond software, 2016). Each trial began with a white fixation cross presented in the centre of the screen for 500 ms. Immediately afterwards, a pair of pictures were presented for 2000 ms, one picture on the left of the screen and the other on the right, 60 mm apart. After this, the pictures disappeared, and a probe – an 'X' – appeared in the position of one of the images. Participants were required to respond to whether the probe appeared in the position of the left or right image, by pressing the 'E' or 'I' key, respectively. The inter-trial interval was 500 ms.

The task consisted of 108 trials. Participants first completed ten practice trials in which neutral picture pairs (images of office supplies) were presented. The main task consisted of two buffer trials (neutral picture pairs) followed by 96 critical trials. Each of the 24 picture pairs were presented four times, both images in each pair were presented twice on the left and twice on the right side of the screen, with the probe appearing an equal number of times behind each image. The visual probe replaced both images in the pair with equal frequency. Trials were presented in a random order for each participant. Eye-movements were recorded during the 2000 ms of stimulus presentation using an eye-tracker (Applied Science Laboratories Eye-Trac D6, Bedford MA) at a sampling rate of 120 Hz. The outcome measure was fixation duration (in milliseconds). Gaze direction bias and reaction time to probes were also measured on each trial and are reported in Appendix B. See Figure 4.1 for a procedural overview of the visual probe task.

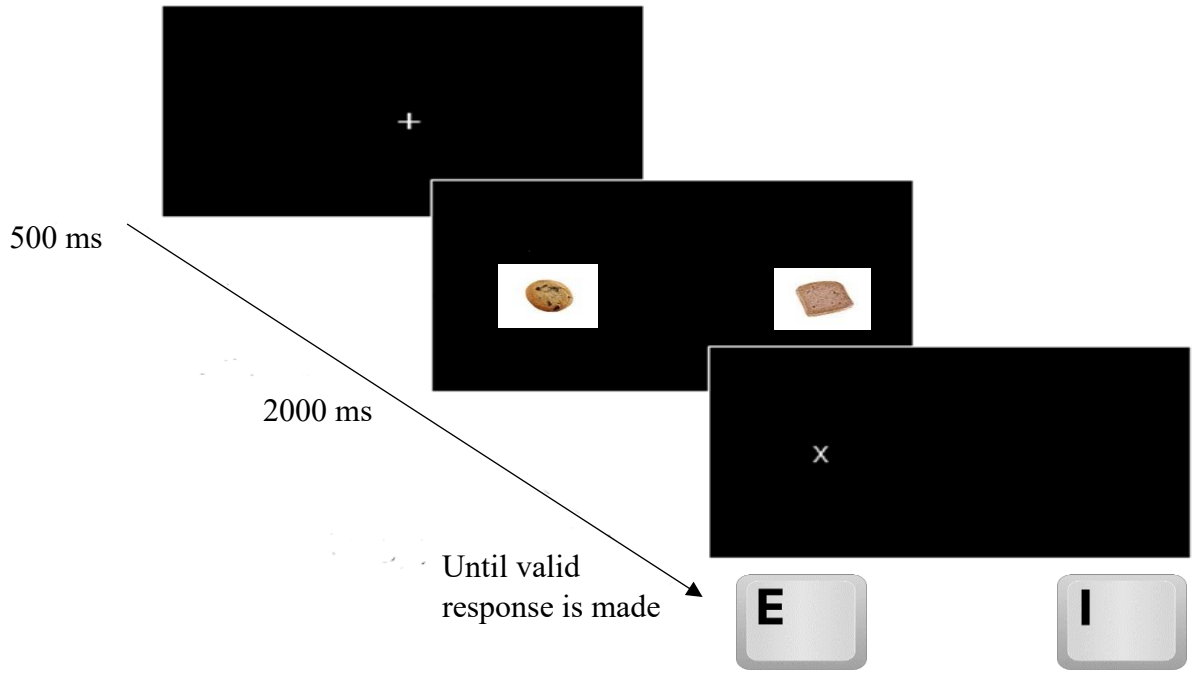

Figure 4.1. Procedural overview of visual probe task with timings.

Salivation: Consistent with previous studies (Brunstrom et al., 2004; Hardman et al., 2014), volume of salivation was measured by participants placing a 3.5 cm dental roll under their tongue for 30 seconds. The dental roll was weighed before and afterwards. This difference in weight (g) was recorded as the amount of salivation.

*Taste-test preparation***:** The taste-test consisted of a 200 g serving of Maryland chocolate chip cookies (487 kcal/100 g) and a 200 g serving of plain tortilla chips (499 kcal/100 g), which were served with 400 grams of water. The foods were served in two identical white bowls. Tortilla chips and cookies were broken into smaller pieces so that participants could not easily monitor the amount consumed (Higgs & Woodward, 2009).

Dutch Eating Behavior Questionnaire (DEBQ; van Strien et al., 1986): The same as in Study 1. The three subscales are restraint (ωt = .92), emotional eating (ωt = .95), and external eating (ωt = .86).

Timeline Follow Back: The same as in Study 1.

Alcohol Use Disorders Identification Test: The same as in Study 1 (ωt = .85).

Snack Urge Scale: The same as in Study 1. However, a composite snack urge score was calculated by adding scores from the four scales, which was then summed across the two snack foods, creating a total score of 800.

Appetite Ratings: The same as in Study 1.

4.4.2.4. Procedure

Test sessions took place between 12:00 and 18:00 on weekdays in the Department of Psychology on the University of Liverpool campus. Each session lasted no longer than 90 minutes. All participants completed both sessions at least one week apart from each other. Participants were told that the present study was investigating how different doses of alcohol can affect reaction times towards and taste perception of food. Participants were told that across both sessions, they would consume two alcoholic drinks: one ‘low’ and one ‘high’ in alcohol. This was done in an attempt to match the anticipated effects of alcohol across conditions. Upon arrival, participants gave written informed consent. As participants were required to consume a light meal an hour before the beginning of the sessions, they next reported when they had last eaten and what they had consumed to ensure they had complied with this instruction. Participants were then breathalysed (all had a BrAC of 0.00) and completed a medical history questionnaire to check for food allergies. Height and weight measurements were then taken in order to calculate the alcohol dosage. Next, baseline salivation was

measured, followed by completion of baseline appetite, snack urge ratings, the DEBQ, AUDIT, and TLFB. Participants then consumed the test drink within ten minutes. This was immediately followed by a ten-minute absorption period where participants sat quietly. Next, the second set of breathalyser, salivation, appetite, and snack urge measures were taken. Participants then completed the VPT. Immediately afterwards, a third salivation measure was taken, which was measured when the taste-test foods were placed in front of the participant (this was the food-exposure measure). The third set of appetite, and snack urge ratings were taken also in the presence of the test foods. Participants then completed the taste test for 15 minutes. During this period, participants were asked to taste the test food as much or as little as they wanted, and to provide ratings based on certain characteristics of the foods (data on ratings were not analysed). Afterwards, a third breathalyser measure was taken, followed by the fourth set of appetite and snack urge ratings. For session 2 only, participants then completed an awareness check, whereby participants were asked to state what they believed to be the true aims of the experiment and were fully debriefed and reimbursed for their time. See Figure 4.2 for a flow chart of the study procedure.

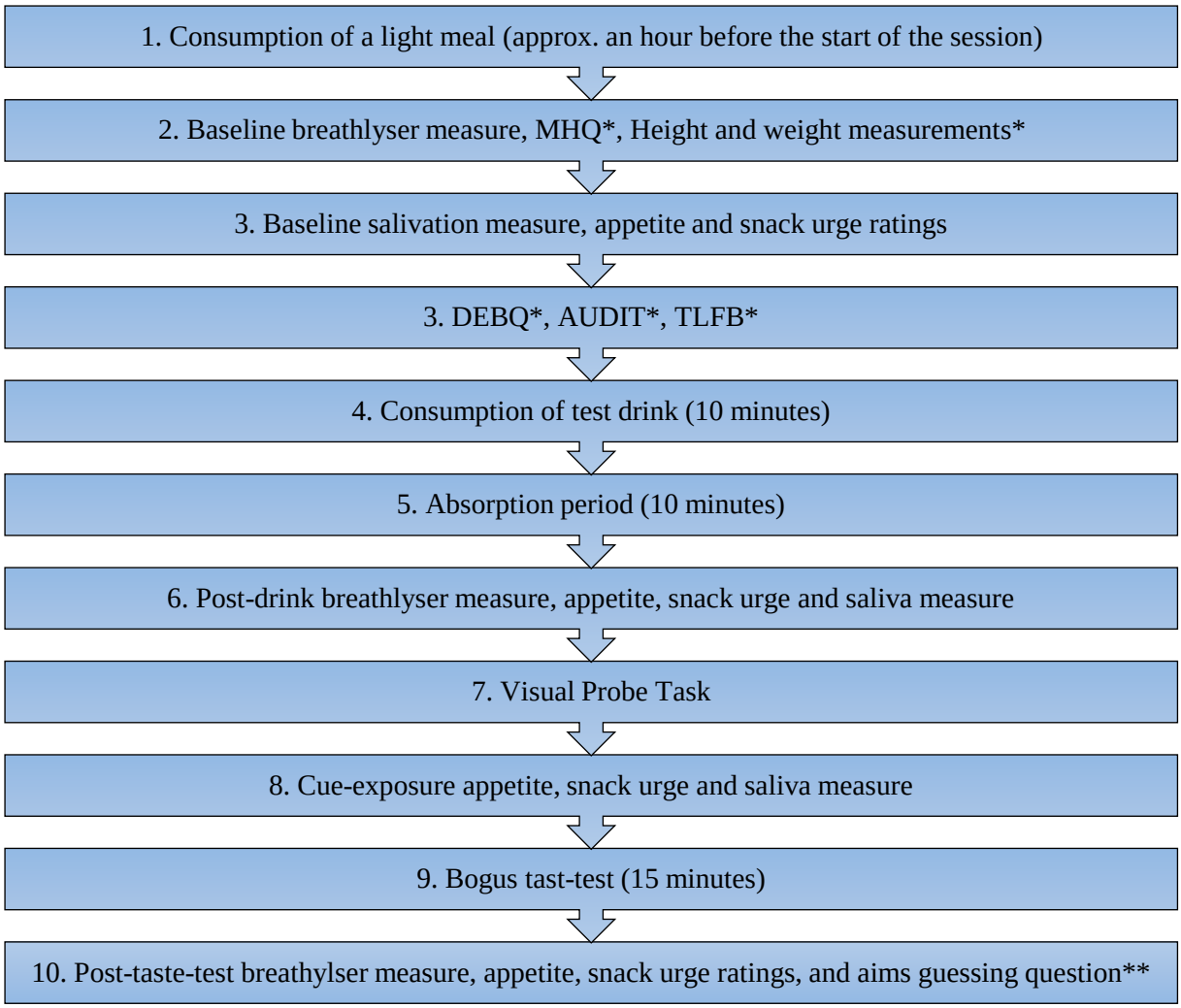

Figure 4.2. Schematic overview of the procedure for Study 3

Note. * = Session 1 only; ** = Session 2 only.

4.4.2.5. Data reduction and analysis

For the eye-tracking data, valid fixations were defined as a stable eye-movement within one degree of a visual angle for 100 ms or longer, as defined in previous research (Jones et al., 2012). Mean bias scores were the primary outcome measure of the eye-tracking data. To calculate mean bias scores, mean fixation duration on control images was subtracted from mean fixation duration on target images; positive scores were indicative of an AB towards target images. Target images were palatable foods in the palatable vs. unpalatable and palatable vs. control trials, and unpalatable foods in the unpalatable vs. control trials. Internal reliability (calculated using McDonalds ω) was calculated for each pair of images (the target image and its matched control image). This was done by calculating the mean fixation duration for each target stimuli and its matched control. The control fixation

duration was subtracted from the target fixation duration. As there were eight image pairs within each pair type, McDonalds ω reflects internal consistency across eight AB scores for each pair type – see Appendix B for these internal reliability scores. Eye-tracking data from four participants were removed from all eye-tracking analyses due to insufficient calibration quality of the eye-tracker, leaving 40 participants for these analyses.

For mean bias scores, a 2 (drink; alcoholic drink, placebo-alcohol) x 3 (pair; palatable vs. control, unpalatable vs. control, palatable vs. unpalatable) repeated measures ANOVA was conducted. One-sample t-tests were also conducted to see whether mean bias scores significantly differed from zero (indicative of bias towards a stimulus type). In order to test whether AB performance was related to appetitive motivational states, we tested whether average food-related AB (on palatable vs control trials) across the two drink conditions correlated with average post-drink snack urge ratings across the two conditions.

To test whether the drink type affected self-report measures of food reward, 2 (drink; alcoholic drink, placebo-alcohol) x 4 (baseline, post-drink, food-exposure, post-taste-test) repeated measure ANOVAs were conducted on snack urge and appetite ratings. Similarly, a 2 (drink; alcoholic drink, placebo-alcohol) x 3 (baseline, post-drink, food-exposure) repeated measures ANOVA was conducted on the measure of salivary response. Pairwise comparisons using Bonferroni correction was conducted when breaking down significant main effects. Greenhouse-Geisser corrected tests are reported where sphericity is violated.

Lastly, paired sample t-tests were conducted to determine whether food intake significantly differed across conditions, and also whether total calories consumed (food intake and drink calories combined) significantly differed across conditions. Finally, using the MEMORE macro for SPSS (Montoya & Hayes, 2017), a moderation analysis was performed to see whether DEBQ restraint scores moderated the effect of drink type on food intake.

4.4.3. Results

4.4.3.1. Participant characteristics

Participant characteristics are shown in Table 4.1. Independent sample t-tests revealed that females had significantly higher DEBQ restraint scores than men. There were no other sex differences for any other participant characteristics in Table 4.1.

Table 4.1. Mean (±SD) for participant characteristics split by sex.

Measure	Male (N = 22)	Female (N = 22)	Total sample (N = 44)
Age (years)	25.64 ± 8.77	25.45 ± 7.84	25.55 ± 8.22
BMI (kg/m^2)	26.00 ± 5.75	25.95 ± 5.85	25.98 ± 5.73
DEBQ Restraint	2.35 ± 0.66*	2.75 ± 0.62*	2.55 ± 0.67
DEBQ Emotional	2.41 ± 0.69	2.51 ± 0.94	2.46 ± 0.81
DEBQ External	3.20 ± 0.48	3.37 ± 0.60	3.29 ± 0.55
AUDIT (out of 40)	10.18 ± 4.35	11.59 ± 5.23	10.89 ± 4.81
7-Day TLFB (in units)	21.45 ± 16.23	17.91 ± 9.81	19.68 ± 13.37

AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index; DEBQ = Dutch Eating Behaviour Questionnaire; TLFB = Timeline Follow-back. * $p = .043$

4.4.3.2. Mean attentional bias scores (Figure 4.3)

As shown in Figure 3.2, there were no significant main effects of drink $F(1, 39) = 0.36$, $p = .551$, $\eta_p^2 = .01$, or pair type $F(1.24, 48.41) = 1.42$, $p = .246$, $\eta_p^2 = .04$, and no significant drink by pair interaction $F(1.24, 48.41) = 0.80$, $p = .400$, $\eta_p^2 = .02$. One-sample t-tests revealed that mean bias scores were significantly greater than zero on the palatable vs. control trials in both the alcohol $t(39) = 3.14$, $p = .003$, $d = 0.50$ and placebo condition $t(39) = 3.14$, $p = .003$, $d = 0.50$, and for unpalatable/control trials in the placebo condition $t(39) = 2.41$, $p = .021$, $d = 0.38$, but not for any other trial type or for either drink condition. There was no significant correlation between average post-drink snack urge ratings and average mean bias scores for palatable vs control trials $r = -.001$, $p = .993$. See Figure 4.3 for mean bias scores for each pair comparison, split by drink condition.

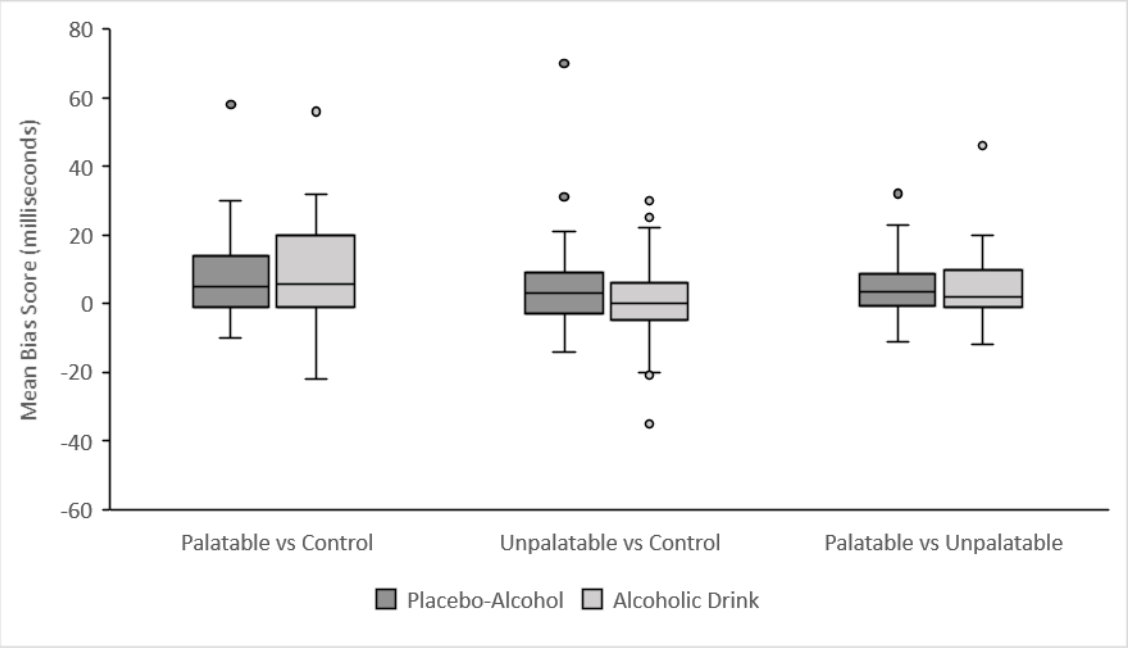

Figure 4.3. Boxplot displaying mean attentional bias scores split by pair type and drink condition. Positive scores indicate greater fixation duration towards palatable images for palatable vs control trials and palatable vs unpalatable trials. Positive scores indicate greater fixation duration towards unpalatable images for unpalatable vs control trials. Dots indicate outliers.

4.4.3.3. Appetite Ratings (Figure 4.4a)

There was a significant main effect of time on appetite ratings $F(2.15, 90.36) = 47.25$, $p < .001$, $\eta_p^2 = .53$ (see Figure 4.4a for comparisons across time points). However, there was no main effect of condition $F(1,42) = 1.20$, $p = .279$, $\eta_p^2 = .03$ or interaction between time and condition $F(2.45, 103.09) = 1.22$, $p = .305$, $\eta_p^2 = .03$.

4.4.3.4. Snack Urge Ratings (Figure 4.4b).

The analysis revealed a main effect of time $F(2.03, 87.26) = 23.34$, $p < .001$, $\eta_p^2 = .35$ (see Figure 4.4b for comparisons across time points). However the main effect of condition $F(1, 43) = 0.31$, $p = .583$, $\eta_p^2 = .01$, and interaction between time and condition $F(2.49, 107.24) = 1.50$, $p = .224$, $\eta_p^2 = .03$ were both non-significant.

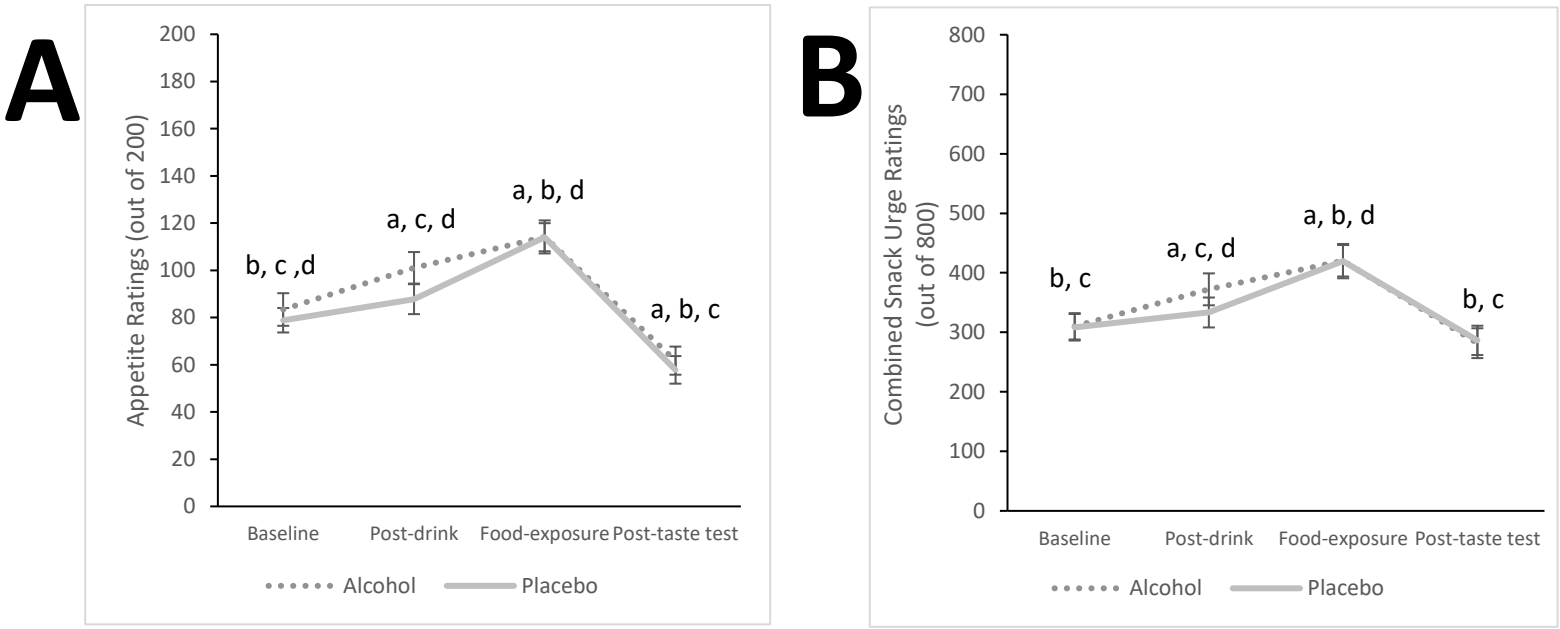

Figure 4.4. Appetite (4.4a) and Snack Urge ratings (4.4b) over time, by condition (Mean ± SEM). Letters refer to Bonferroni corrected pairwise comparisons breaking down significant differences ($p < .05$) between time points: a = difference from baseline; b = difference from post-drink; c = difference from food exposure, d = difference from post-taste test.

4.4.3.5. Salivation Measure

There was a significant main effect of time $F(2, 86) = 6.56$, $p = .002$, $\eta_p^2 = .13$. Pairwise comparisons revealed that the amount of salivation was lower at baseline than at post-drink ($p = .018$; mean difference = 0.05; 95% CI [-0.09, -0.01]) and at food exposure ($p = .005$; mean difference = 0.07; 95% CI [-0.12, -0.02]). However, there was no significant difference between post-drink and food exposure ($p = 1.00$; mean difference = 0.02; 95% CI [-0.03, 0.07]). The main effect of condition, $F(1, 43) = 0.54$, $p = .468$, $\eta_p^2 = .01$, and the time by condition interaction $F(1.72, 74.14) = 0.38$, $p = .655$, $\eta_p^2 = .01$ were both non-significant.

4.4.3.6. Calorie Measures (Figure 4.5)

Mean and standard deviation for calories consumed from the test drink were: alcohol (153.22 ± 37.06); placebo (3.03 ± 0.73). The range of test drink volume (millilitres) was 205.63 – 541.82.

Paired samples t-tests revealed no significant difference between conditions for the amount of food calories consumed during the taste test, $t(43) = -0.92$, $p = .361$, $d = .14$. However there was a significant difference in total calories consumed (drink calories combined with food calories) $t(43) = 3.37$, $p = .002$, $d = 0.51$, with participants in the alcohol condition consuming significantly more calories overall relative to the placebo condition. See Figure 4.5 for caloric intake split by drink condition. The difference in food caloric intake and total caloric intake between drink conditions was compared between sex. Findings revealed no sex differences in the amount of food calories consumed between drink conditions $t(34.80) = 1.81$, $p = .079$, $d = 0.54$ or in the amount of total calories consumed between drink conditions $t(33.40) = 1.36$, $p = .183$, $d = 0.41$. Lastly, the moderation analysis revealed that DEBQ restraint scores did not moderate the effect of drink type on food intake $b = 87.23$ [-15.45, 189.91], $SE = 50.88$, $t(42) = 1.71$, $p = .094$.

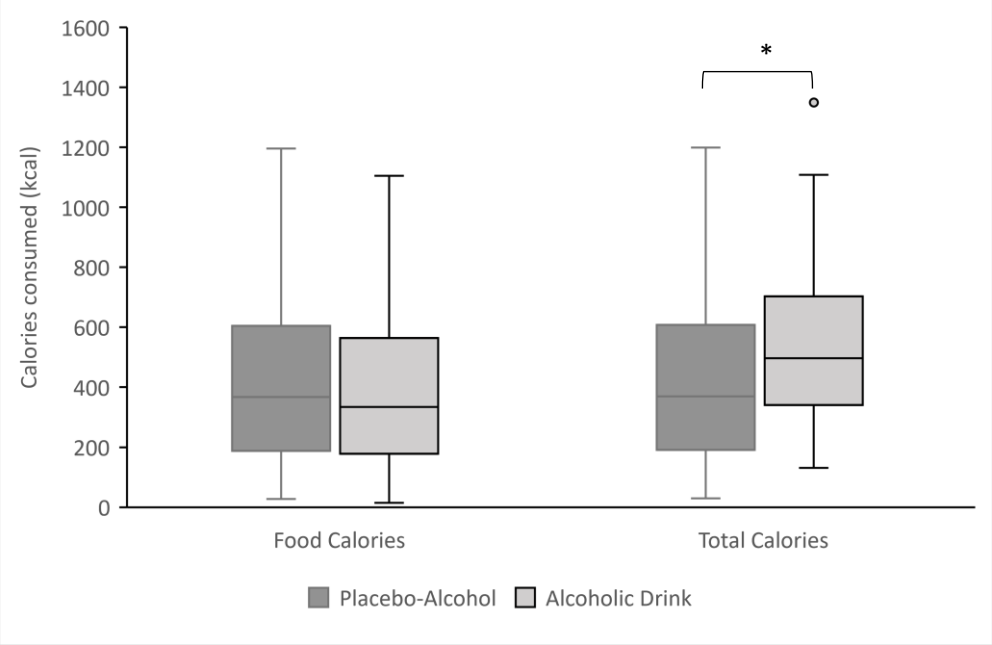

Figure 4.5. Boxplot displaying number of calories consumed during the *ad libitum* taste test (food calories) and combined with calories consumed from the test drink (total calories), split by condition. Note. *$p = .002$. Dots indicate outliers.

4.5. Interim Discussion

Study 3 investigated whether an alcohol dose of 0.3 g/kg could alter food-related attentional biases, self-report appetitive motivational states, salivation response, and food intake relative to a

placebo-alcohol. The results showed that alcohol consumption did not produce greater attentional biases towards food-related stimuli, nor was there evidence of alcohol-induced changes in appetitive motivational states or increase in salivation towards palatable foods. Although null findings, these results are in line with predictions of Field et al. (2016) who suggest that changes in AB are, in part, the result of changes in motivational states. Furthermore, Study 3 showed no change in *ad libitum* food intake. However, total caloric intake was significantly greater in the alcohol condition relative to placebo. This latter finding is in line with previous research which has consistently shown that the calories within an alcoholic beverage appears to be additive and are not compensated for at a later eating episode (Caton et al., 2004; Christiansen et al., 2016; Kwok et al., 2019; Rose et al., 2015; Yeomans et al., 1999).

These null findings may be explained by the dose of alcohol being too low to produce meaningful changes in appetitive motivational states and food intake. Previous research has found that a dosage of 0.6 g/kg produces significant changes in snack urge ratings and food intake (Christiansen et al., 2016; Rose et al., 2015). Furthermore, Rose and Duka (2006) found that self-report appetitive motivation towards alcohol increased after a dose of 0.6 g/kg but not 0.3 g/kg, relative to placebo. A higher alcohol dosage was not used in Study 3 because other research has found an AB towards other types of appetitive stimuli at a dose of 0.3 g/kg (Schoenmakers et al., 2008). Furthermore, higher doses of alcohol have consistently failed to enhance alcohol-related AB (0.6 g/kg - Duka & Townshend, 2004; 0.65 g/kg - Monem & Fillmore, 2019), despite evidence showing increases in alcohol craving at similar doses (Duka et al., 1999; Rose & Duka, 2006). These null findings may be because higher doses of alcohol are problematic for measuring AB due to oculomotor impairments following alcohol consumption (Abroms et al., 2006; Moser et al., 1998; Rohrbaugh et al., 1988). Therefore, an AB task which uses ocular behaviour (i.e., eye movements) as its outcome measure may mask an effect of AB when using higher doses of alcohol, despite enhancements in food-related appetitive motivational states.

4.6. Study 4

4.6.1. Overview

Study 4 investigated whether consumption of a 0.6 g/kg dose of alcohol can enhance AB towards images of food, increase self-report measures of food reward (appetite and snack urge ratings) and increase food intake, relative to a placebo-alcohol. Additionally, in order to provide a further manipulation check between drink conditions, the study tested whether consumption of the alcoholic drink could produce an increase in alcohol-related motivational states (alcohol urge ratings) and AB towards alcohol cues, as this dose has previously been shown to increase motivation for alcohol (Duka et al., 1999; Rose & Duka, 2006).

In order to mitigate the issue of impairments in ocular behaviours at higher doses, Study 4 measured both food and alcohol-related AB with a pictorial modified Stroop task, which captures AB using manual response latencies rather than ocular fixation behaviour. The pictorial form of the Stroop task has been shown to produce acceptable levels of internal reliability (Ataya et al., 2012).

An additional aim was to examine the role of top-down and bottom-up processes in driving alcohol-induced increases in food intake. Dual-process models argue that eating behaviour is determined by an interaction of bottom-up drives relating to motivational orientation and food reward, and top-down cognitive control (Appelhans, 2009). Several studies have demonstrated the combined effect of food reward and impulsivity in predicting eating behaviour and weight change (Appelhans et al., 2011; Kakoschke et al., 2015; Nederkoorn et al., 2009; Nederkoorn et al., 2010; Price et al., 2015; Rollins et al., 2010). For example, Nederkoorn et al. (2009) showed that poor response inhibition (a type of impulsivity) was related to overeating only when desire to eat was also high. It is therefore possible that top-down control and bottom-up reward processes interact to facilitate alcohol-induced overeating. To test this, Study 4 investigated whether trait impulsivity (specifically motor impulsivity) and alcohol-induced changes in food-related AB (using the pictorial modified Stroop task) could interactively predict changes in food intake across drink conditions.

It was predicted there would be an enhanced food and alcohol-related AB after consumption of the alcoholic drink compared with a placebo-alcohol. We also predicted that participants would

consume more calories in an *ad libitum* taste test after consumption of the alcoholic drink, and that appetite, snack urge and alcohol urge ratings would increase to a greater extent after alcohol consumption compared with the placebo. We predicted a positive correlation between post-drink snack urge ratings and food-related AB, and between post-drink alcohol urge ratings and alcohol-related AB. Lastly, we predicted that the interaction term of motor impulsivity and change in food-related AB between conditions would significantly predict change in food intake between conditions.

4.6.2. Method

4.6.2.1. Participants

The study was powered based on an earlier version of the meta-analysis by Hardman et al. (2020) (Hardman et al., 2018) which found a correlation of $r = 0.14$ between food-related AB and food craving. Based on 80% power and an alpha level of 5%, 81 participants would be needed in order to detect the same effect size between drink conditions. 84 participants (male = 13) aged between 18 and 26 y (M = 18.75; SD = 1.13) completed both sessions in order to counterbalance the order of drink condition and the order of target and neutral blocks in the Stroop task (see measures section for further details). Six additional participants completed session one, but did not return for session 2, and were therefore excluded from all analyses. Participants were recruited through the university undergraduate credit scheme. Inclusion criteria was the same as in Study 3 with the following changes: participants were able to take part if they wore glasses to correct their vision, but participants were excluded if they were colour-blind. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee (reference number: 5529). Participants were reimbursed through course credits. The method and analysis strategy for this study were pre-registered on the Open Science Framework (https://osf.io/cnaxr/).

4.6.2.2. Design

The study used a single-blind randomised within-subjects design with drink type (alcoholic drink, placebo-alcohol) as the independent variable. Each participant completed both conditions in

two sessions separated by at least one week. The order of drink condition was randomised and counterbalanced across participants.

4.6.2.3. Measures

Beverage Preparation and Administration: This was the same as in Study 3 with the following changes: the alcohol dose was at 0.6 g/kg (5.35 UK units of alcohol for a participant weighing 70 kg); participants consumed the test drink in three separate portions, each served in set 5-minute intervals, meaning that participants consumed the test drink in 15 minutes.

Modified Stroop Task: Participants completed four blocks of a pictorial modified Stroop task on PsychoPy2 (Peirce et al., 2019). Each block consisted of 40 trials: ten different images, presented four times, each time with a different coloured border surrounding the image (either blue, red, yellow, or green). The four blocks consisted of: food images (five images of cookies and five of tortilla chips), food control images (five of drink coasters and five of leaves), alcohol images (alcoholic drinks), and alcohol control images (office stationery). The food and food control images were the same as in Study 3 with the addition of two extra pairs (sourced from the same website as in Study 1). Alcohol and alcohol control images were taken from a previous study (Field et al., 2011).

Each image was 351 x 259 pixels and was surrounded by a 10-pixel coloured border. Images were matched on visual properties such as colour and brightness. For each trial, participants were required to respond to the colour of the border surrounding the image as quickly and as accurately as possible, participants did so by providing a key response (d, f, j, and k). The keys were marked with coloured stickers that matched the corresponding colours for responses. The same colours were matched with the same key for every participant. Participants were instructed to place the index and middle finger of the left hand on the ‘d’ and ‘f’ key respectively, and the same fingers of the right hand on the ‘j’ and ‘k’ key.

In both sessions, participants completed a block of 40 practice trials using filler images (a plain image surrounded by each border colour 10 times) before the main task in order to become familiar with the location of each key response. Participants were required to repeat the practice block

until they provided correct responses on at least 95% of trials within this block. The main task consisted of four blocks, this was completed in blocked presentation in order to avoid any interference carry over effects (Waters et al., 2003). The order of blocks was counterbalanced such that for the first session, half of the participants saw the presentation in the following order: food images, food control images, alcohol images, alcohol control images. The other half of participants saw the presentation in the following order: food control images, food images, alcohol control images, alcohol images. All participants saw the other presentation order in their second session.

For the main task each trial began with a fixation cross, presented in the middle of the screen for 500 ms. Following this, the image was presented in the middle of the screen until a response was made. The inter-trial interval was 500 ms. There was also a 5-second inter-block break.

Barratt Impulsivity Scale (BIS-11; Patton et al., 1995): Trait impulsivity was assessed across three dimensions; attentional (ωt = .77), motor (ωt = .71), and non-planning (ωt = .80). The BIS consists of 30 items (score Rarely/Never – Almost always/Always) with higher scores indicating greater impulsivity. The motor dimension (motor impulsivity) of the scale (which captures acting without thinking) was measured to see whether it predicts alcohol-induced change in food intake. Data on the other dimensions of the BIS were recorded to characterise the sample.

Alcohol Urge Questionnaire (AUQ; Bohn et al., 1995): Participants were asked to provide current alcohol urge ratings across three domains: desire for alcohol; expectation of positive effect from drinking; and inability to avoid drinking if alcohol was available. Items were responded to on a scale from 1 to 7 with high scores being indicative of greater alcohol urge.

Subjective intoxication scales (SIS; Duka et al., 1998): Participants were asked to provide subjective feelings of being ‘lightheaded’, ‘irritable’, ‘stimulated’, ‘alert’, ‘relaxed’, and ‘contented’ before and after consumption of the test drink. Data for scores on lightheaded ratings are reported in the results section as acute alcohol consumption has shown to increase lightheaded feelings when measured within the context of food intake (Caton et al., 2004; Caton et al., 2005; Westerterp-Plantenga et al., 1999). The remain data are presented in Appendix B.

Unit estimation: Participants were asked how many units of alcohol they believed they had consumed at the end of each session to see whether participants believed that the placebo-alcohol contained alcohol.

The following measures were used as in Study 3: DEBQ (restraint ωt = .96; emotional eating ωt = .96; external eating ωt = .92); TLFB; AUDIT (ωt = .74), snack urge scale, appetite ratings, taste-test.

4.6.2.4. Procedure

The procedure was similar to that of Study 3 with the following changes: the cover story was changed such that participants in Study 4 were told that the aims were to see how different doses of alcohol can affect visual and taste perception of food. At the beginning of the session, participants completed baseline alcohol urge and subjective intoxication scale ratings and completed the BIS; participants completed a 20-minute absorption period after consumption of their test drink; participants completed post-drink alcohol urge and subjective intoxication scale ratings; after completing the taste test, participants provided a set of alcohol urge ratings and were asked how many units of alcohol they believed the test drink contained. For the second session, the procedure was identical to session 1, apart from participants consumed the other drink type, and also completed the modified Stroop task in the other block order, and did not complete height and weight measures, the DEBQ, BIS, TLFB, or AUDIT. Lastly, at the end of the second session, participants completed the aims awareness question and were fully debriefed and reimbursed. See Figure 4.6 for a schematic overview of the procedure.

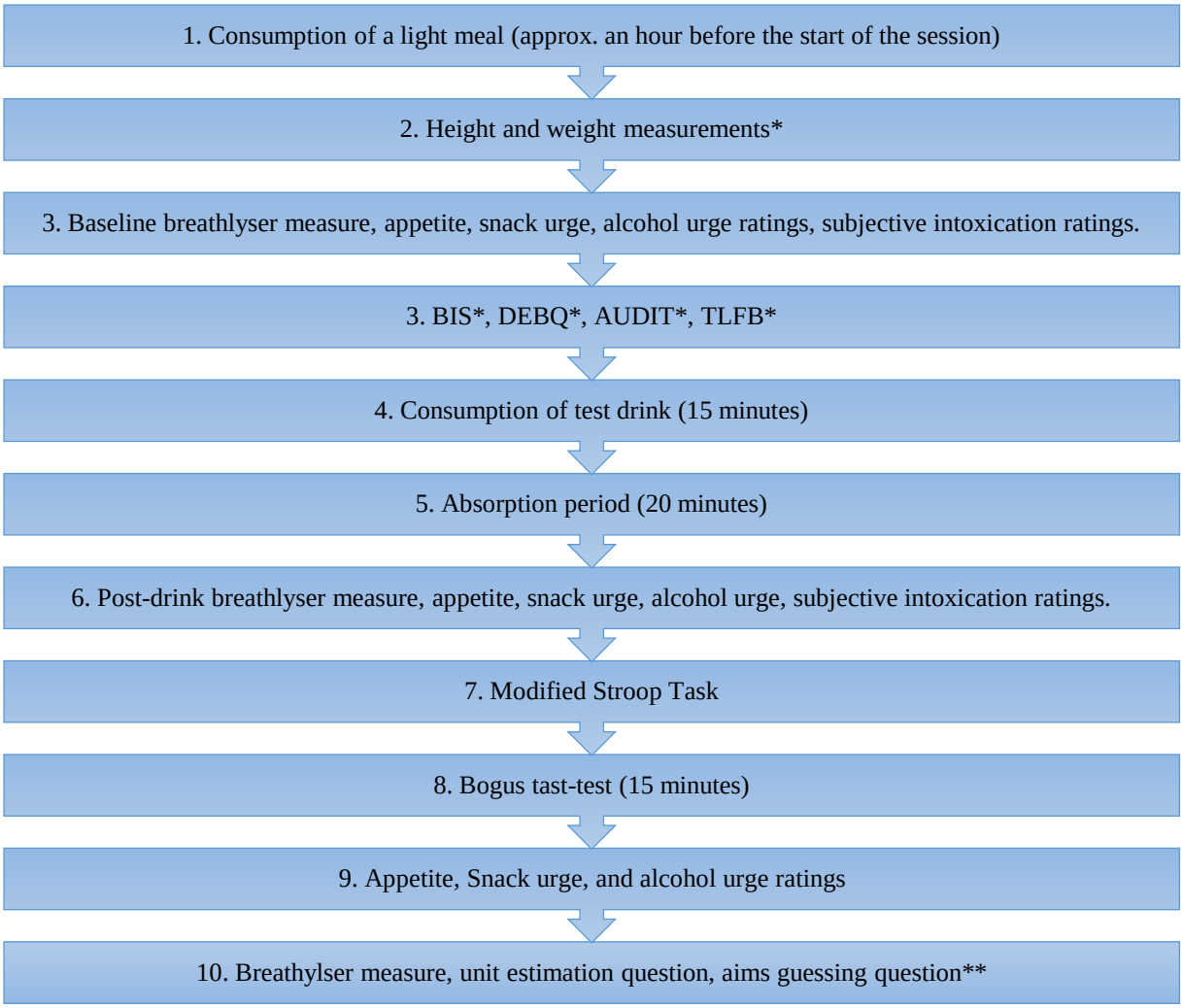

Figure 4.6. Schematic overview of the procedure for Study 4. Note. * = Session 1 only; ** = Session 2 only.

4.6.2.5. Data reduction and analysis

Before AB scores were calculated, all responses which were quicker than 200 ms, slower than 2000 ms, three standard deviations above the individual mean response, and incorrect, were removed. This resulted in the removal of 4.98% of trials. After data reduction, mean reaction time on control trials was subtracted from mean reaction time on target trials - positive scores were indicative of an AB towards the target stimuli (food images and alcohol images). Internal reliability (calculated using McDonalds ω) was calculated for each pair of stimuli (the target image and its matched control image). This was done by calculating the mean reaction time across all coloured borders for each target stimuli and its matched control. The control reaction time was subtracted from the target reaction time. As there were 10 pairs for both the food-related and alcohol-related AB, McDonalds ω

for each AB type reflects internal consistency across 10 AB scores. See Appendix B for these internal reliability scores.

A 2 (drink; alcoholic drink, placebo-alcohol) x 2 (task; food AB, alcohol AB) repeated measures ANOVA was conducted on AB scores. Follow-up paired samples t-tests were conducted in order to investigate the effect of the drink condition on AB score, separately for the two types of target stimuli. One-sample t-tests were conducted for both AB tasks, split by drink, in order to test whether mean bias scores significantly differed from zero. In order to test whether AB performance was related to appetitive motivational states, two correlations were conducted to test whether average food-related AB across the two drink conditions correlated with average post-drink snack urge ratings across the two conditions, and to test whether average alcohol-related AB correlated with average alcohol urge ratings at post-drink, between the two drink conditions.

Two paired samples t-tests were conducted to examine whether food intake and total caloric intake (food and drink calories combined) differed between drink conditions. A moderation analysis was performed to see whether DEBQ restraint scores moderated the effect of drink type on food intake.

Separate 2 (drink; alcoholic drink, placebo-alcohol) x 3 (baseline, post-drink, post-taste test) repeated measure ANOVAs were conducted for appetite ratings, total snack urge ratings, and total alcohol urge ratings (Greenhouse-Geisser corrected tests are reported where sphericity is violated). A one-sample t-test was conducted to see whether the estimated number of units consumed in the placebo condition significantly differed from zero to confirm whether participants believed there to be alcohol in this condition. A 2 (drink; alcoholic drink, placebo-alcohol) x 2 (baseline, post-drink) repeated measures ANOVA was conducted for subjective intoxication scale scores (see results section for analysis of lightheaded scores and Appendix B for analysis of all other subjective intoxication scale scores).

A hierarchical regression was conducted with food AB scores in the placebo condition entered in step 1 as a control variable. Change in food-related AB between conditions (positive scores

indicating a greater AB in the alcohol condition relative to placebo) and trait motor impulsivity and the interaction between change in food-related AB by trait motor impulsivity were entered as predictor variables at step 2. Change in food intake between conditions (positive scores indicative of greater food intake in the alcohol condition relative to placebo) was the dependent variable. Due to high VIF scores (> 10), the predictor variables were mean centred. This reduced VIF scores to an acceptable level.

4.6.3. Results

4.6.3.1. Participant characteristics

Participant characteristics are shown in Table 4.2. A series of Independent samples t-test revealed a sex difference in BMI and DEBQ restraint scores between males and females, with females having a significantly greater BMI and restraint score, relative to males.

Table 4.2. Sample characteristics split by sex (mean ± SD).

Measure	Male (N = 13)	Female (N = 71)	Total sample (N = 84)
Age (years)	19.23 ± 2.24	18.66 ± 0.77	18.75 ± 1.13
BMI (kg/m^2)	20.50 ± 1.84*	22.76 ± 3.68*	22.41 ± 3.54
DEBQ Restraint	1.62 ± 0.77*	2.36 ± 0.94*	2.25 ± 0.95
DEBQ Emotional	2.43 ± 0.93	2.87 ± 0.88	2.80 ± 0.89
DEBQ External	3.32 ± 0.58	3.34 ± 0.68	3.34 ± 0.66
AUDIT (out of 40)	13.92 ± 3.86	13.15 ± 4.40	13.27 ± 4.31
7-Day TLFB (in alcohol units)	22.35 ± 17.08	17.49 ± 8.98	18.24 ± 4.31
BIS (attentional)	18.38 ± 2.72	17.68 ± 4.02	17.79 ± 3.85
BIS (motor)	21.85 ± 3.53	22.86 ± 4.21	22.70 ± 4.11
BIS (non-planning)	24.62 ± 4.68	24.70 ± 4.63	24.69 ± 4.61
BIS (total)	64.85 ± 6.67	65.24 ± 10.31	65.18 ± 9.81

AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index; DEBQ = Dutch Eating Behaviour Questionnaire; TLFB = Timeline Follow-back; BIS = Barratt Impulsivity Scale.
*p < .05

4.6.3.2. Modified Stroop (Figure 4.7)

There was a nonsignificant main effect of task on mean bias scores $F(1, 83) = 0.46$, $p = .501$, $\eta_p^2 = .01$, a nonsignificant main effect of drink on mean bias scores $F(1, 83) = 0.44$, $p = .437$, $\eta_p^2 = .01$, but a significant task by drink interaction $F(1, 83) = 4.62$, $p = .034$, $\eta_p^2 = .05$. Follow-up paired samples t-tests revealed that mean bias scores for the alcohol AB measure did not differ between

drink conditions $t(83) = 1.05$, $p = .297$, $d = 0.08$. However, mean bias scores on the food AB task were significantly greater in the alcohol condition relative to the placebo condition $t(83) = 2.28$, $p = .025$, $d = 0.18$. One-sample t-tests revealed that mean bias scores in the food AB task in the alcohol condition were significantly greater than zero $t(83) = 3.33$, $p < .001$, $d = 0.36$, but scores in the placebo-alcohol condition did not differ from zero $t(83) = 0.54$, $p = .593$, $d = 0.06$. For the alcohol AB task, mean bias scores did not differ from zero in the alcohol condition $t(83) = 0.42$, $p = .679$, $d = 0.05$, but were significantly greater than zero in the placebo condition $t(83) = 2.01$, $p = .047$, $d = 0.22$. There were no significant correlations between average post-drink alcohol urge scores and average alcohol-related AB ($r = .133$, $p = .229$) or between average post-drink snack urge scores and average food-related AB scores ($r = -.065$, $p = .558$). See Figure 4.7 for mean bias scores for each attentional bias task, separately for each drink condition.

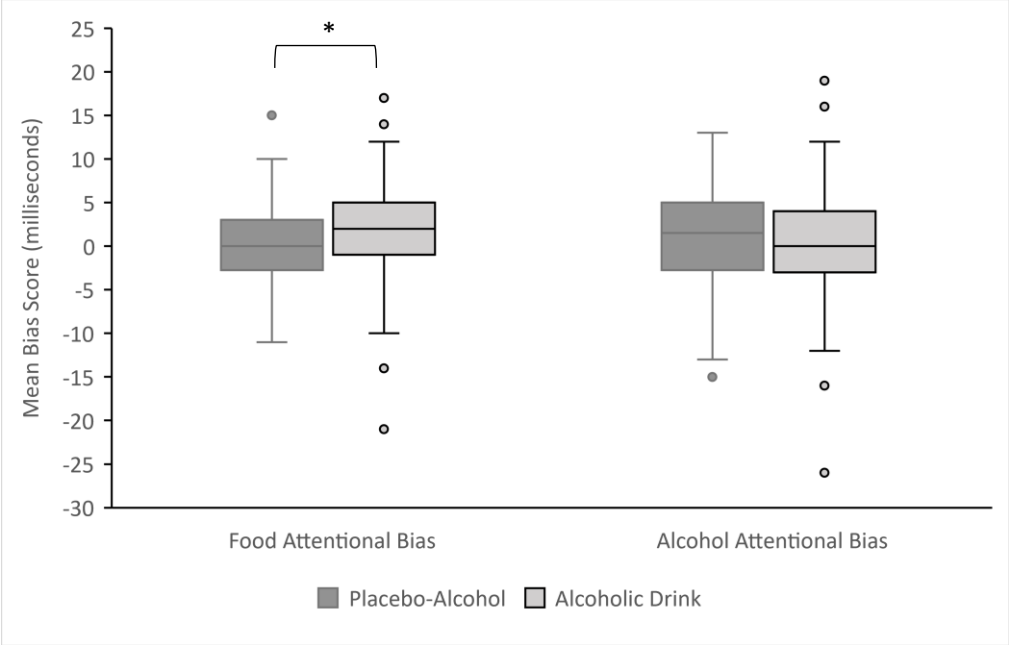

Figure 4.7. Boxplot displaying mean bias scores split by AB task and drink condition. Note: * $p = .025$. For food attentional bias, positive scores indicate greater fixation duration towards food images relative to control images. For alcohol attentional bias, positive scores indicate greater fixation duration towards alcohol images relative to control images. Dots indicate outliers.

4.6.3.3. Caloric Intake (Figure 4.8)

Mean and standard deviation for calories consumed from the test drink were: alcohol (249.07 ± 43.68); placebo (4.92 ± 0.86). The range of test drink volume (millilitres) was 372.10 – 800. There

was a greater number of calories consumed from the taste test in the alcohol condition compared with the placebo condition $t(83) = 4.67$, $p < .001$, $d = 0.36$. Similarly, there was greater total caloric intake in the alcohol condition compared with the placebo condition $t(83) = 15.11$, $p < .001$, $d = 1.17$. The moderation analysis revealed that DEBQ restraint scores did not moderate the effect of drink type on food intake $b = -15.03$ [-57.85, 27.80], $SE = 21.53$, $t(82) = 0.70$, $p = .487$. See Figure 4.8 for caloric intake split by drink condition.

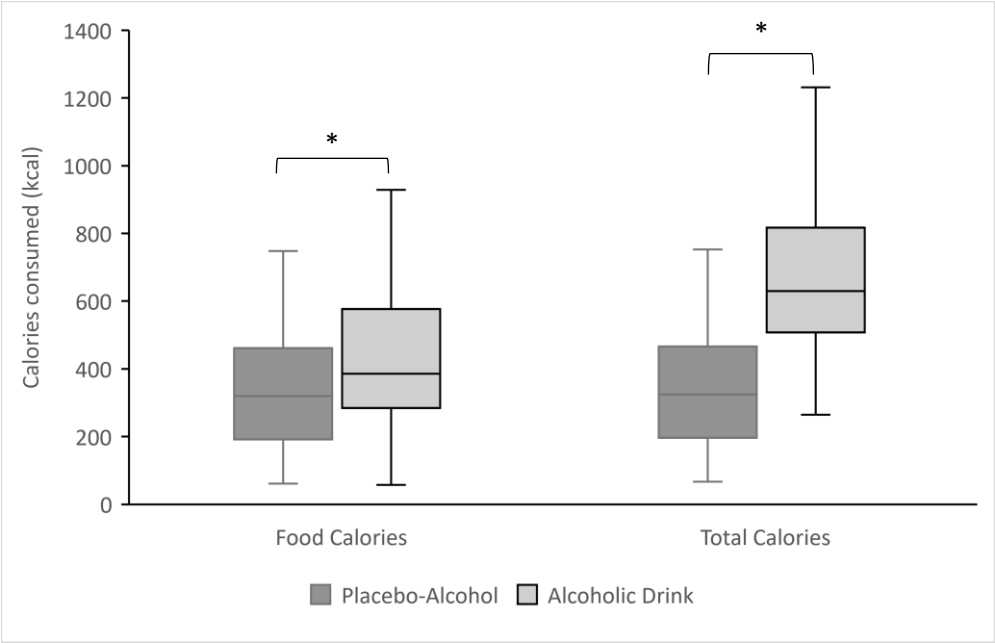

Figure 4.8. Boxplot displaying number of calories consumed during the *ad libitum* taste test (food calories) and combined with calories consumed from the test drink (total calories), split by condition Note: * $p < .001$. Dots indicate outliers.

4.6.3.4. Appetite Ratings (Figure 4.9a)

There was a significant main effect of drink on appetite ratings $F(1, 83) = 5.67$, $p = .019$, $\eta_p^2 = .06$, with consumption of the alcoholic drink producing greater appetite ratings. There was also a significant main effect of time $F(1.79, 148.80) = 45.50$, $p < .001$, $\eta_p^2 = .35$. Pairwise comparisons revealed that baseline appetite ratings were significantly lower than post-drink ratings ($p < .001$; mean difference = 23.67; 95% CI [-32.04, -15.31]) but were significantly greater than post-taste test ratings ($p = .013$; mean difference = 13.21; 95% CI [2.17, 24.25]). Post-drink ratings were significantly greater than post-taste test ratings ($p < .001$; mean difference = 36.89; 95% CI [27.77, 46.01]). Lastly,

there was no significant drink by time interaction $F(2,166) = 0.75$, $p = .474$, $\eta_p^2 = .01$. See Figure 4.9a for appetite ratings across each time point, split by condition.

4.6.3.5. Snack Urge Ratings (Figure 4.9b)

There was a main effect of drink $F(1, 83) = 10.54$, $p = .002$, $\eta_p^2 = .11$, with those in the alcohol condition reporting greater snack urge. There was also a significant main effect of time $F(1.46, 121.54) = 13.13$, $p < .001$, $\eta_p^2 = .14$, and a significant drink by time interaction $F(1.85, 153.49) = 7.08$, $p = .002$, $\eta_p^2 = .08$. A follow-up repeated measures ANOVA comparing difference scores for snack urge ratings between drink conditions for each time point revealed that the difference in snack urge ratings between drink conditions was greater at post-drink compared with baseline ($p < .001$; mean difference = 59.20; 95% CI [26.25, 92.16]), however there was no significant difference between baseline and post-taste test difference scores ($p = .098$; mean difference = 37.56; 95% CI [-4.66, 79.78]) nor between post-drink and post-taste test difference scores ($p = .599$; mean difference = 21.64; 95% CI [-19.25, 62.54]). See Figure 4.9b for snack urge ratings across each time point, split by condition.

Alcohol Urge Ratings (Figure 4.9c)

There was a significant main effect of drink $F(1, 83) = 31.63$, $p < .001$, $\eta_p^2 = .28$, with significantly greater alcohol urge ratings in the alcohol condition. There was also a significant main effect of time $F(1.82, 32.79) = 30.85$, $p < .001$, $\eta_p^2 = .27$ and a significant drink by time interaction $F(2, 166) = 21.00$, $p < .001$, $\eta_p^2 = .20$. A follow-up repeated measures ANOVA comparing differences between drink conditions across the three time points revealed that the difference in alcohol urge ratings between drink conditions was significantly lower at baseline compared with post-drink ($p < .001$; mean difference = 5.00; 95% CI [2.44, 7.56]) and post-taste test ($p < .001$; mean difference = 6.02; 95% CI [3.56, 8.49]). However, there was no difference between post-drink and post-taste test difference scores ($p = .811$; mean difference = 1.02; 95% CI [-3.28, 1.23]). See Figure 4.9c for alcohol urge ratings across each time point, split by condition.

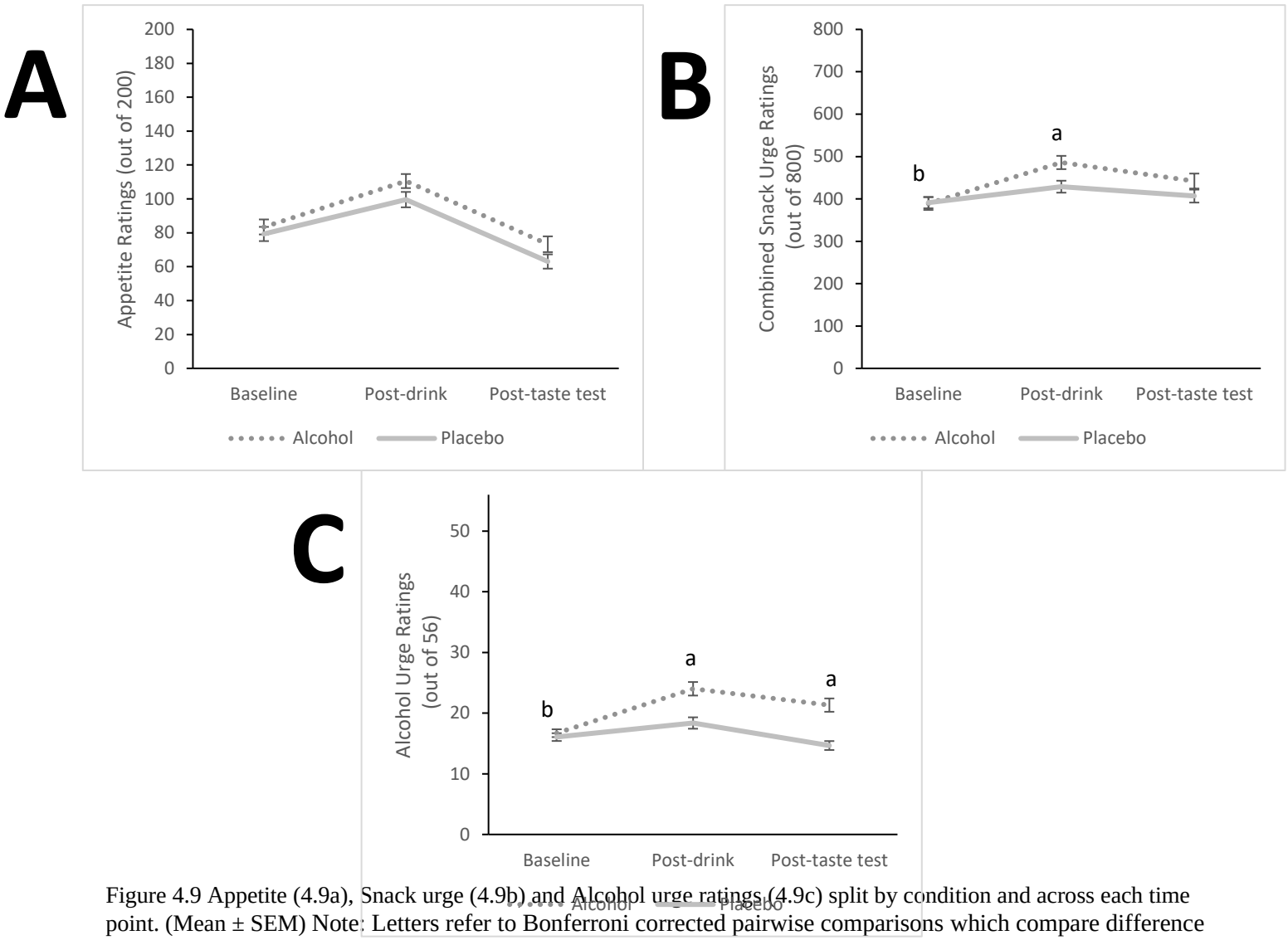

Figure 4.9 Appetite (4.9a), Snack urge (4.9b) and Alcohol urge ratings (4.9c) split by condition and across each time point. (Mean ± SEM) Note: Letters refer to Bonferroni corrected pairwise comparisons which compare difference scores between drink conditions across each time point ($p < .05$): a = different from baseline difference scores; b = different from post-drink difference scores; c = different from post-taste test difference scores.

4.6.3.6. Lightheaded ratings (Figure 4.10) and Unit Estimation

A one-sample t-test revealed that the number of units estimated to be in the placebo drink was significantly greater than zero $t(83) = 11.55$, $p < .001$, $d = 1.26$. A 2 (drink; alcohol, placebo) x 2 (baseline, post-drink) repeated measures ANOVA was conducted on lightheaded scores. There was a significant main effect of drink on these scores $F(1,83) = 119.46$, $p < .001$, $\eta_p^2 = .59$, with those in the alcohol condition providing greater scores. There was also a main effect of time $F(1,83) = 150.59$, $p < .001$, $\eta_p^2 = .65$, with greater levels at post-drink. There was also a significant drink x time interaction $F(1,83) = 34.07$, $p < .001$, $\eta_p^2 = .58$. This was due to a nonsignificant difference between drink conditions at baseline $t(83) = .000$, $p = 1.00$, $d = 0.00$, but a significant difference at post-drink $t(83) = 11.70$, $p < .001$, $d = 0.91$. See Figure 4.10 for lightheaded scores across time points, split by drink condition.

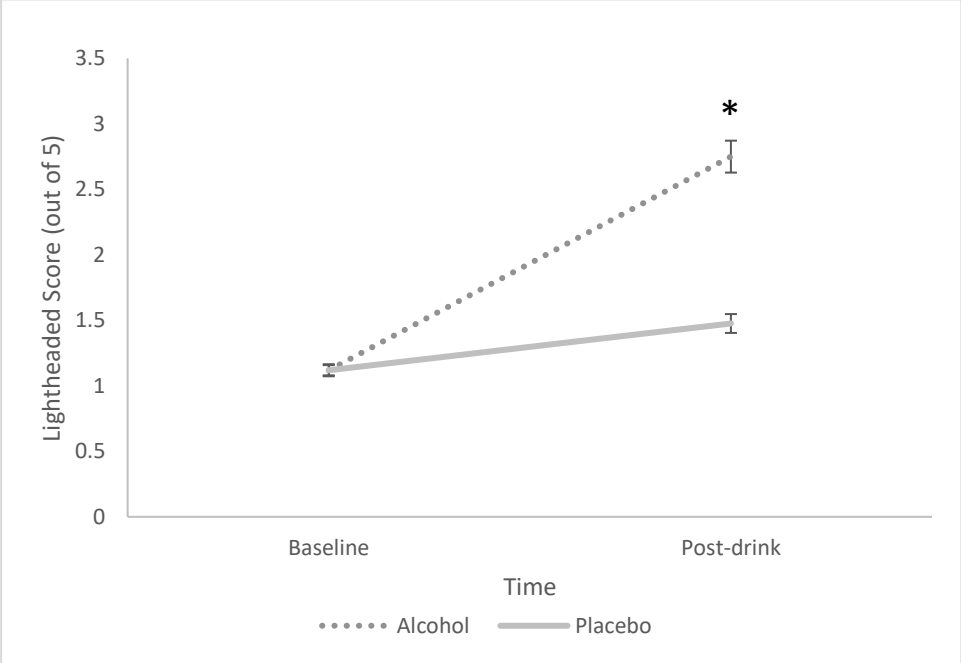

Figure 4.10. Lightheaded scores split by drink condition and time point. (Mean ± SEM) * *p < .001*

4.6.3.7. Predictors of change in food intake

The regression analysis was performed to test whether motor impulsivity, differences in food-related AB between conditions, and the interaction between them, predicted change in food intake

across the two conditions. The regression model predicted 1% of variance in change in food intake, adjusted R^2 = .01, $F(4, 79) = 1.28$, p = .287. There were no significant predictor variables in the model: change in food-related AB (β = -.13, p = .351); motor impulsivity (β = -.17, p = .142); change in food-related AB by motor impulsivity (β < .01, p = .989).

4.7. General Discussion

Collectively, findings from Studies 3 and 4 substantially differ. Study 3 failed to show any alcohol-induced increases relating to both implicit and explicit measures of food reward and food intake. Conversely, in Study 4, alcohol consumption enhanced snack urge ratings, food-related AB and food intake, along with increases in alcohol urge ratings. Taken together, findings from both studies suggests that alcohol intoxication increases appetitive motivational states, food-related AB and food intake, but only when administered above a certain dose (in this case 0.6 g/kg). This seemingly dose-dependent response is in line with previous research by Caton et al. (2004), who demonstrated that food intake was significantly greater after consumption of 4 UK units of alcohol compared with consumption of 1 UK unit. Results from the explicit measures of food reward are consistent with other research which has shown that an alcohol dose of 0.6 g/kg is sufficient to increase snack urge ratings (Rose et al., 2015). Food intake also significantly increased after alcohol consumption, which has been demonstrated in several studies (see Kwok et al., 2019 for review). Dietary restraint did not moderate this effect, suggesting that those with higher levels of dietary restraint (when measured using the DEBQ) are not more susceptible to alcohol-induced increase in food intake. This is in line with previous research which has also failed to demonstrate that restrained individuals are more susceptible to alcohol-induced overeating (Christiansen et al., 2016a; Poppitt et al., 1996; Ouwens et al., 2003). However, as this was a secondary analysis, our study was not specifically powered to test for moderation by dietary restraint. Therefore these findings need to be treated with caution.

The food-related AB findings in Study 4 reveal that in contrast to previous research (Monem & Fillmore, 2019), alcohol intoxication can increase the magnitude of food-related AB. This discrepancy in findings may be explained by the use of a different AB task. As mentioned, the null finding of Monem and Fillmore (2019) may have been due to alcohol-induced impairments to visual

performance, as their measure of AB used concurrent eye-tracking. Impairments to the ocular system are more pronounced at higher doses of alcohol (Abroms et al., 2006: Rohrbaugh et al., 1988). The Stroop task used in the current study did not use ocular behaviour as its outcome measure, and may therefore have been better suited to the current dose and allowed an AB effect to be detected. However, this suggestion remains speculative and further research should elucidate whether such an effect is dependent on the type of AB measure used.

Study 4 failed to show an alcohol-induced increase in alcohol-related AB. Although unexpected, this finding is in line with previous studies which have shown that alcohol consumption fails to enhance AB towards alcohol cues at doses of 0.6 g/kg (Duka & Townshend, 2004) or 0.65 g/kg (Monem & Fillmore, 2019), but does increase self-reported urge to drink (e.g. Rose & Duka, 2006 – 0.6 g/kg). Overall, this suggests that alcohol consumption increases appetitive motivation for alcohol, but that different assessment procedures may be focusing on different aspects of motivation and/or value towards certain stimuli.

Relatedly, the present findings raise questions regarding the construct validity of AB in the context of food reward. Theories suggest that AB is, in part, indicative of appetitive motivational states (Field et al., 2016). However, there was no significant correlation between measures of food motivational state (snack urge ratings) and AB in either study. This null finding is likely due to insufficient statistical power as we were unable to detect a small correlational effect – findings from a recent meta-analysis has shown the association between food cravings and food-related AB to be r = 0.13 (Hardman et al., 2020). Nevertheless, the present findings suggest that AB should not be used as an index of food reward in isolation. Future research which aims to measure changes in AB should do so alongside other measures of food-related motivational states.

Contrary to our prediction, in Study 4 there was no interaction between motor impulsivity and change in food-related AB as a predictor of change in food intake. This finding does not support a dual-process model of eating behaviour within the context of acute alcohol consumption, which predicts that overeating is determined by an interaction of bottom-up (food reward responsivity) and top-down (impulsivity) processes. One explanation for this null finding could be due to alcohol

intoxication in itself impairing state components of impulsivity at similar doses to those used in the present study (Christiansen et al., 2016; Fillmore & Vogel-Sprott, 1999; Mulvihill et al., 1997). Therefore, the predictive power of trait motor impulsivity may have been masked by alcohol-induced changes in state behaviours (i.e., after alcohol consumption, impulsive behaviours increased and therefore may have become level across all participants within this condition). Therefore, future studies may wish to investigate if alcohol-induced changes in state impulsivity interact with food reward to predict changes in food intake.

There were some limitations with the current studies. Firstly, in Study 3, palatability of the food-related stimuli in the attentional bias task was categorised as palatable/unpalatable based on its nutritional composition – palatable food being high in fat and/or sugar and unpalatable food being low in these components, as previously defined (Rogers & Brunstrom, 2016). However, it is possible that participants did not perceive these stimuli categorically as palatable or unpalatable. Therefore, future research should measure palatability of these stimuli categories to check whether the manipulation in stimuli type is successful. Secondly, the two studies were not perfectly matched on all methodological components. For example, Study 4 implemented an absorption period double the length to Study 3. This was done to avoid participants feeling satiated after consumption of the test drink, as the volume of liquid consumed in Study 4 was greater due to the implementation of a larger alcohol dose. Another methodological difference was the type of AB measure used. This was changed because, as previously mentioned, it was more appropriate to use response latency rather than ocular attention as the outcome measure when implementing a higher alcohol dose. A third limitation is that Study 4 did not test an equal number of males and females. This may be problematic if alcohol affects food intake differently in males and females, however a recent meta-analysis has shown that alcohol-induced increases in eating occurs in both males and females (Kwok et al., 2019). Finally, the alcoholic and caloric content of the test drinks were not matched across participants. It could be argued that because the caloric content in the alcoholic drink was greater than in the placebo, appetite levels across conditions may have differed. However, data from both studies show that appetite ratings were not suppressed by greater caloric intake from the test drink, suggesting that this difference in caloric

intake did not affect findings. Instead, the alcohol dose was adjusted by bodyweight in order to achieve a better matched breath alcohol concentration across participants. This is important because evidence from the present studies and previous research (e.g., Caton et al., 2004) suggest that an alcohol-induced effect on eating behaviour is dependent on the dosage of alcohol. Therefore, it was essential that participants received a dose which produced a more consistent breath alcohol concentration across participants. If the alcohol dose was unadjusted, some participants may not have received a dosage high enough to produce changes in behaviour.

In summary, the two studies revealed that alcohol's ability to affect indices of food reward appears to be dose-dependent - at lower doses of alcohol consumption, changes to appetitive motivational states appear to be minimal. However, both Studies 3 and 4 found an alcohol-induced increase in total caloric intake, which may increase the risk of weight gain if these calories are not compensated for. Greater doses of alcohol consumption significantly increased food-related AB, motivational states, and food intake. This adds to the continuingly growing body of evidence which demonstrates that acute alcohol consumption alters behavioural states relevant to eating behaviour, and further implicates drinking behaviour as an important risk factor for weight gain.

Chapter 5: Investigating the effect of trait impulsivity and alcohol consumption on change in BMI

5.1. Overview

Findings from Chapter 3 revealed that trait motor impulsivity did not interact with change in food-related attentional bias to predict change in food intake, suggesting that at least when using these measures, a dual-process account of eating behaviour does not explain alcohol-induced changes to food intake. Thus far, this thesis has focused on the effects on acute food intake. However, Chapter 4 investigates whether longer-term changes to drinking behaviour can predict changes to BMI. Specifically, over the course of 12 months, drinking behaviour was used as a predictor variable of change in BMI. Furthermore, Chapter 4 also investigated whether trait motor impulsivity moderated any effect of change in drinking behaviour on change in BMI, to determine whether individuals with higher levels of impulsivity are more susceptible to alcohol-induced weight gain.

5.2. Abstract

Alcohol consumption has been implicated as a risk factor for weight gain. However, the longitudinal temporal relationship between drinking behaviour and weight gain is unclear. The present study investigated the effect of drinking behaviour (measured separately as drinking frequency and drinking intensity of an episode) on BMI at 3 time points over the course of 12 months in a sample of first year undergraduate students (N = 374). It also investigated whether baseline motor impulsivity interacted with change in drinking behaviour at 6 months and 12 months to predict change in BMI at 6 months and 12 months, respectively. First year undergraduate students completed an online survey and recorded self-report BMI and compensatory behaviours (physical activity and compensatory eating in response to alcohol consumption) at the beginning of the academic year (baseline) and at 6 and 12-months later. Firstly, a longitudinal cross-lagged model was used to test for the associations between drinking behaviour and BMI at the 3 time points, after controlling for gender. Findings revealed that neither type of drinking behaviour predicted BMI at a subsequent time point. Next, using a hierarchical regression analysis, after controlling for confounding variables, the interaction of motor

impulsivity (taken at baseline only) and change in drinking behaviour between baseline and 6 months and between baseline and 12 months did not predict change in BMI between these respective times. Collectively, these findings suggest that drinking behaviour does not significantly predict subsequent BMI in a sample of first-year undergraduate students. This adds to the inconsistent findings regarding the relationship between alcohol consumption and adiposity.

5.3. Introduction

The transition to university is a period where students experience significant changes to their environment and lifestyle. Research has consistently shown that University students gain a significant amount of weight during their first year of University, with meta-analyses showing this increase to be on average 5 pounds (Vadeboncoeur et al., 2015; Vella-Zarb & Elgar, 2009). Several variables have been shown to predict weight change in freshman (first year) students, including trait disinhibition and binge eating behaviours (Finlayson et al., 2012), decrease in physical activity (Butler et al., 2004), high levels of perceived stress (Serlachius et al., 2007), and high levels of unhealthy food consumption (Levitsky et al., 2004).

Alcohol consumption is another lifestyle factor which changes when transitioning to University. Drinking behaviour among undergraduate students is high, with approximately two-thirds of students from Ireland and the UK classified as displaying harmful drinking (Davoren et al., 2016). Furthermore, alcohol consumption of undergraduates has consistently been shown to be greater than their non-attending counterparts (Johnston et al., 2015; Johnston et al., 2016; Kypri et al., 2005). Given that previous evidence has implicated excessive alcohol consumption as a risk factor for weight gain (Sayon-Orea et al., 2011), university students who engage in excessive alcohol consumption may be at risk of undesirable weight gain.

However, the relationship between drinking behaviour and weight-related outcomes within the general population is not linear, but appears to be J-shaped. Non-drinkers show lower levels of adiposity compared with light-to-moderate drinkers, whereas heavier drinkers show the most elevated levels (Arif & Rohrer, 2005; Duvigneaud et al., 2007; Lukasiewicz et al., 2005; Tolstrup et al., 2005; Wakabayashi, 2010; Wannamethee et al., 2005). Furthermore, several studies have found a positive

association between drinking behaviour and measures of adiposity in heavy drinkers (Halkjær et al., 2006; MacInnis et al., 2014; Rissanen et al., 1991; Sayon-Orea et al., 2011; Schütze et al., 2009; Wannamethee et al., 2004). A meta-analysis by Sayon-Orea et al. (2011) showed that a positive association exists between alcohol consumption and body weight for those who display heavy drinking patterns but not for moderate or light alcohol drinkers. Taken together, findings suggest that heavier drinking behaviours produce a greater risk for weight gain, whereas light-to-moderate levels appear not to pose a risk.

Within student samples, the association between drinking behaviour and adiposity is mixed. Some studies have shown a lack of correspondence between drinking behaviour and weight measures (Deliens et al., 2013; Fazzino et al., 2019; Kasparek et al., 2008; Pliner & Saunders, 2008; Pope et al., 2017), whereas others have found this to be a significant determinant of weight change (Adams & Rini, 2007; Bodenlos et al., 2015; de Vos et al., 2015; Deforche et al., 2015; Economos et al., 2008; Lloyd-Richardson et al., 2008; Zagorsky & Smith, 2011). As discussed in Section 1.4.2, this inconsistency of findings may result from differences in how drinking behaviour is operationalised, with evidence suggesting that drinking intensity (how much alcohol is consumed in a typical drinking episode) may be a better predictor of BMI compared with drinking frequency (how frequently a person consumes alcohol).

The role of compensatory behaviours may also partially explain inconsistent findings. Levels of physical activity have been shown to positively correlate with alcohol consumption (Piazza-Gardner & Barry, 2012; Conroy et al., 2015). Another example of compensatory behaviour is through caloric restriction on planned drinking days, this behaviour has been found in a proportion of undergraduates ranging from 14% - 46% (Burke et al., 2010; Giles et al., 2009; Roosen & Mills, 2015).

Individual differences regarding the effect of alcohol consumption on weight gain may exist between students, namely individuals with greater levels of impulsivity may be more susceptible to alcohol-induced weight gain. Dual-process models of eating behaviour postulate that overeating results from an interaction of top-down and bottom-up processes (Appelhans, 2009). Multiple studies

have demonstrated that the interaction of these processes can predict eating behaviour and weight change (Appelhans et al., 2011; Kakoschke et al., 2015; Meule & Platte, 2016; Nederkoorn et al., 2010; Rollins et al., 2010). Given that acute alcohol consumption has been shown to alter bottom-up processes (such as increasing food reward) (Caton et al., 2004; Rose et al., 2015; Schrieks et al., 2015), we may expect individuals with high levels of impulsivity to be particularly susceptible to alcohol-induced overeating, and therefore alcohol-induced weight gain. This is because individuals with higher levels of impulsivity may find it more difficult to inhibit an eating response after an alcohol-induced increase in food reward. Preliminary support of this suggestion comes from a study which demonstrated that performance on a Go/No-Go-Task (a measure of impulsivity) and change in alcohol consumption interactively predicted weight loss, whereby greater reductions in alcohol consumption predicted greater weight loss in individuals with higher levels of impulsivity (Kase et al., 2016). One interpretation of these findings is that weight loss occurred after a reduction in alcohol consumption more so for these individuals because when these individuals do drink, they are unable to inhibit prepotent responses (such as the initiation of food intake) and are therefore more susceptible to overeating after alcohol consumption. However, Study 4 failed to show that trait motor impulsivity interacted with change in food-related attentional bias to predict change in food intake, suggesting that short-term alcohol-induced food intake is not predicted by trait impulsivity and changes in attentional bias. Nevertheless, this suggestion remains untested within the context of change to BMI over time.

The present study investigated whether change in alcohol consumption predicts BMI change in a sample of first-year UK Undergraduate students. This was achieved through completion of an online survey during the first year of university at three time points: the beginning of the academic year, and 6 and 12 months after this point. The study measured drinking behaviour separately as drinking frequency (how often alcohol is consumed) and drinking intensity (how many units an individual consumes in a typical drinking episode). The study also controlled for compensatory behaviours – physical activity and compensatory eating and behaviours in response to alcohol consumption.

It was hypothesised that drinking behaviour (specifically drinking intensity but not drinking frequency) would predict BMI and that change in drinking behaviour and the interaction term of motor impulsivity and change in drinking behaviour (drinking intensity but not frequency) would predict an increase in BMI.

5.4. Method

5.4.1. Participants

In total, 803 respondents opened the survey. After data cleaning (see data collection and cleaning section), 374 respondents completed Wave 1, 121 respondents completed both Wave 1 and 2, and 62 respondents completed all three waves. See Figure 5.1 for overview of respondents at each wave. First-year students from any University in the UK were recruited through online advertisements, social media, and word-of-mouth. Respondents could only participate if they: were aged 18 or 19 (to increase the likelihood that respondents were transitioning from a non-University setting); were in their first year of University; were consumers of alcohol; had not taken a gap year prior to University. Ethical approval was gained from the University of Liverpool Health and Life Sciences Research Ethics Committee (reference number: 2081).

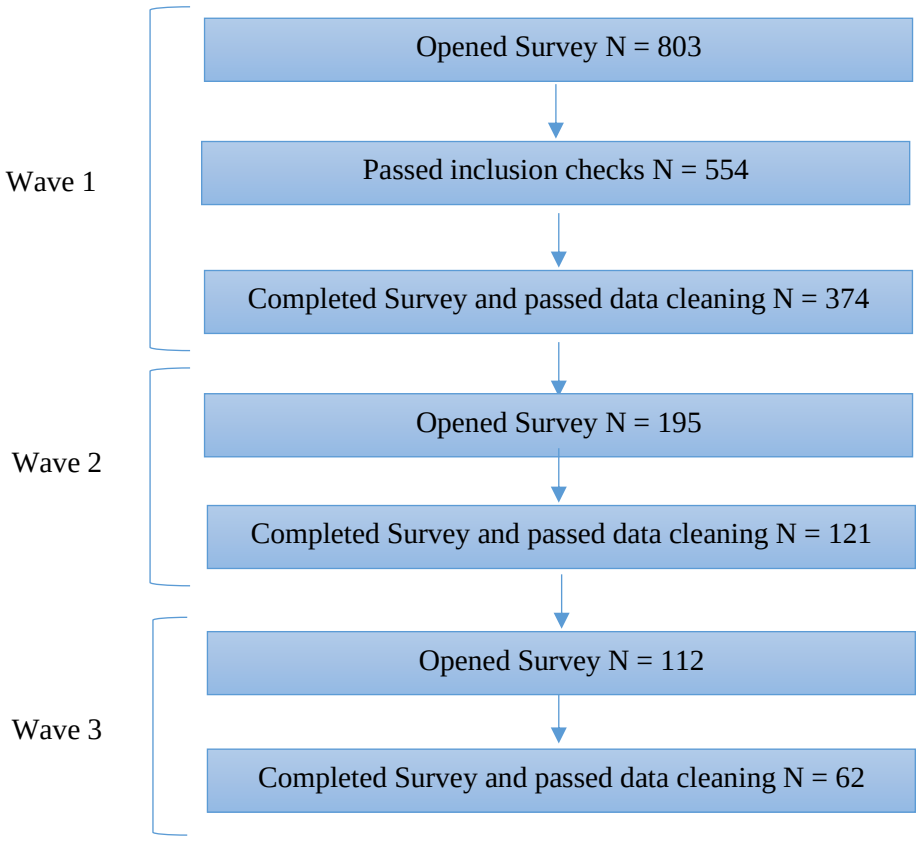

Figure 5.1. Overview of attrition across the three waves.

5.4.2. Measures

Body Mass Index: BMI was calculated by self-reported height and weight at the three time points. Height and weight were excluded if: height was outside of the plausible range of 1.22 – 2.13 m and weight was outside the range of 34 – 227 kg. These cut-offs have been used in previous research (Kersbergen & Robinson, 2019; Noël et al., 2010).

Drinking behaviour: Drinking behaviour was measured using the Alcohol Use Disorders Identification Test—Consumption (AUDIT-C) (Saunders et al., 1993). At each wave, participants were asked to report their drinking behaviour over the past month. Item 1 'How often did you have a drink containing alcohol in the past month?' was used as the measure of drinking frequency. Item 2

‘How many drinks did you have on a typical day when you were drinking in the past month?’ was the measure of drinking intensity.

Physical activity: Physical activity was measured using the International Physical Activity Questionnaire (IPAQ) 7-day short-form (Craig et al., 2003). Participants were asked to report both the frequency (number of days in the last 7 days) and amount of time which they engaged in three levels of exercise: walking, moderate physical activity and vigorous physical activity in a day. The metabolic equivalent of task (MET) of each PA intensity level was multiplied by the duration and the frequency of the PA and was expressed as MET-minutes per week (MET-min/wk). Each MET-min/wk was then summed across the three levels to produce a measure of total physical activity.

Trait Impulsivity (BIS-11; Patton et al., 1995): Trait impulsivity was assessed across three dimensions; attentional (ω = .80), motor (ω = .75), and non-planning (ω = .78) and is described in Section 3.6.2.3. Motor impulsivity was included as a predictor variable because this subscale is positively correlated with BMI (Price et al., 2015; van Koningsbruggen et al., 2013) and has shown to interact with bottom-up process to predict change in BMI (Meule & Platte, 2016). Data on total BIS score and the other dimensions of the BIS were collected and recorded to characterise the sample.

Compensatory Behaviours: Compensatory Behaviours were measured using the Compensatory Eating and Behaviours in Response to Alcohol Consumption Scale (CEBRACS; Rahal et al., 2012). The measure is a 21-item questionnaire which consists of four factors: alcohol effects (restricting food intake to enhance the effect of alcohol), bulimia (reflecting bulimic-like behaviours in response to alcohol consumption), dietary restraint and exercise, restriction (skipping meal or eating less in a day). The sub-scales measure caloric compensatory behaviour before, during, and after consuming alcohol, over the past month. All items were summed to produce a total score.

Food Frequency Questionnaire: The food frequency questionnaire was a shortened version of a previously validated snack intake measure (Inchley et al., 2001) and consists of four questions which measured how often respondents consume savoury snack foods, sweet snack foods, convenience foods, and fast foods/take away foods. Eight options were presented and ranged from ‘Never or less

than once a month' to 'More than 3 times a day, every day'. The score across the four items were summed as a total score (out of 32). The food frequency questionnaire focuses only on consumption of palatable foods as previous research has demonstrated increased preferences and intake of palatable foods (Caton et al., 2004; Schrieks et al., 2015; Christiansen et al., 2016). This score was included to characterise the sample in terms of level of unhealthy food consumption.

5.4.3. Data Collection and Cleaning

Data were collected across three waves, once every 6 months. Data for Wave 1 was collected during September/October of the first academic year. Data for wave 2 was collected in March/April of the first academic year. Data for wave 3 was collected in September/October of the second academic year, capturing change in BMI and drinking behaviours across a 12-month period. The dataset consists of data from two cohorts of students – a cohort who began University in September/October 2017 and a second cohort who began University in September/October 2018. This was done in order to produce a larger sample size.

Participants were excluded from the analysis if they provided responses outside of pre-determined cut-offs for BMI and responses on the IPAQ. For IPAQ responses, total activity greater than 960 minutes per week was considered an outlier and removed from analysis, as is recommended in the IPAQ guidelines (IPAQ Research Committee, 2005).

5.4.4. Procedure

The survey was hosted on Qualtrics. In Wave 1, participants were firstly presented with the information sheet, followed by the consent form. Next, they completed a series of questions to ensure that they were eligible to take part. Eligible participants then stated which type of household they were living in, options were: Self-catered (University), Self-catered (Private), Catered (University), Parental Home. Next, respondents provided details of their height, weight and gender. Next, they completed the AUDIT-C, followed by the IPAQ, CEBRACS, food frequency questionnaire and BIS-11. Wave 2 and 3 were identical to Wave 1, apart from the following changes: participants did not complete the BIS-11 in Wave 2 or 3 (as it is a trait measure), and participants were debriefed in Wave

3. As reimbursement, participants were entered into a prize draw with the chance to win £150 worth of shopping vouchers if they completed all three waves.

5.4.5. Data Analysis

Using AMOS (Version 25), autoregressive cross-lagged models were conducted to examine relationships between drinking behaviour and BMI. The model allows for the investigation between variables X at time point 1 and Y at time point 2, after controlling for the stability paths (e.g., the effect of BMI at Wave 1 on BMI at Wave 2), allowing for cross-lagged associations to predict any residual variance between variables. The models measured whether drinking behaviour at Wave 1 and 2 could predict BMI at Wave 2 and 3, respectively, after gender was controlled for at Wave 2 and 3 BMI. Conversely, BMI at Wave 1 and 2 were tested to see whether they predicted drinking behaviour at time points 2 and 3, respectively. Drinking behaviour was operationalised separately as drinking frequency (model 1; question 1 of the AUDIT-C) and drinking intensity (model 2; question 2 of the AUDIT-C). For both models, gender was included as a control variable on BMI at time 2 and 3. Both models were conducted twice, once as a complete case analysis which included only participants who had completed all 3 waves (N = 62) and once when removing responses from participants who did not complete Wave 2 (N = 121). Missing data were estimated using full information maximum likelihood. Goodness of fit cut-offs for a good fit of the data (taken from Hu & Bentler, 1999) were the following: SRMR ≤ 0.08; CFI ≥ 0.95; RMSEA ≤ 0.06.

Additionally, using SPSS (Version 26), hierarchical regression models were conducted to examine whether the interaction of motor impulsivity and change in drinking behaviour significantly predicts change in BMI. As in the cross-lagged model, drinking behaviour was operationalised separately as drinking frequency (model 3) and drinking intensity (model 4). In Step 1, gender, motor impulsivity, Wave 1 BMI and change scores in IPAQ, CEBRACS, and drinking behaviour was entered into the model. In Step 2, the interaction term of motor impulsivity and change in drinking behaviour was entered. Positive change scores indicate that a score increased in a subsequent wave (e.g., a higher score in Wave 2 compared with Wave 1). Both models were conducted twice - measuring change in BMI between Wave 1 and 2 and measuring change in BMI between Wave 1 and

3. For all regression models, multiple imputation was used to estimate missing data. Specifically, a five-iteration pooled estimate for each regression coefficient was calculated, yielding a pooled R^2 and F-value for each step of the model, as has been done in previous research (e.g., Field et al., 2017).

5.5. Results

5.5.1. Participant Characteristics (Table 5.1)

See Table 5.1 for an overview of participant characteristics. Out of the 374 respondents who completed Wave 1, 45 were in catered university accommodation, 247 were in self-catered university accommodation, 54 were in self-catered private accommodation and 28 lived in a parental home.

Table 5.1. Descriptive statistics of participants split by each wave.

Variable	Wave 1	Wave 2[a]	Wave 3[a]
Gender ratio M:F (% male)	102:272 (27.27%)	N/A	N/A
IPAQ (MET-min/wk)	3399.29 ± 2305.62	3264.72 ± 2085.94	4256.46 ± 2798.61
CEBRACS (out of 105)	29.21 ± 9.36	31.35 ± 11.19	29.31 ± 10.38
BMI (kg/m^2)	22.37 ± 4.24	22.79 ± 4.13	22.37 ± 6.95
Drinking Frequency (out of 4)	2.75 ± 0.84	2.44 ± 0.96	2.21 ± 1.01
Drinking Intensity (out of 5)	1.29 ± 1.03	1.37 ± 0.95	1.22 ± 0.97
Food Frequency Score (out of 32)	13.25 ± 3.56	12.69 ± 3.49	12.55 ± 3.59
BIS Motor	18.66 ± 3.85	N/A	N/A
BIS Total	56.98 ± 9.14	N/A	N/A

[a] Scores of missing data were calculated through a five-iteration multiple imputation pooled estimate.

5.5.2. Cross-lagged model analysis (Tables 5.2, Figures 5.2 and 5.3)

As shown in Figures 5.2 and 5.3, drinking behaviour (regardless of the measure) was not a significant predictor of BMI at a later time point in any of the models. Similarly, BMI was not a significant predictor of drinking behaviour at any time point. As shown in Table 5.2, model 1 was found to be a good fit of the data. Model 2 was shown to be a partially good fit of the data. In both cases, the model fit was driven by the stability paths between drinking behaviour at subsequent time points, and between BMI at subsequent time points.

Table 5.2. Goodness of fit indices for each model, split by analysis type.

	Model 1 – Complete Case Analysis (N = 62)	Model 1 – removal of incomplete Wave 2 responses (N = 121)
$\chi^2(df)$	8.33(10)	10.73/10
p-value	.596	.379
CFI	1.00	1.00
RMSEA	< .01	.03
SRMR	.05	N/A[1]
	Model 2 – Complete Case Analysis (N = 62)	Model 2 – removal of incomplete Wave 2 responses (N = 121)
χ^2/df	16.94/10	18.74/10
p-value	.076	.044
CFI	.95	.97
RMSEA	.11	.09
SRMR	.07	N/A[1]

[1]SRMR cannot be estimated when imputing missing data.

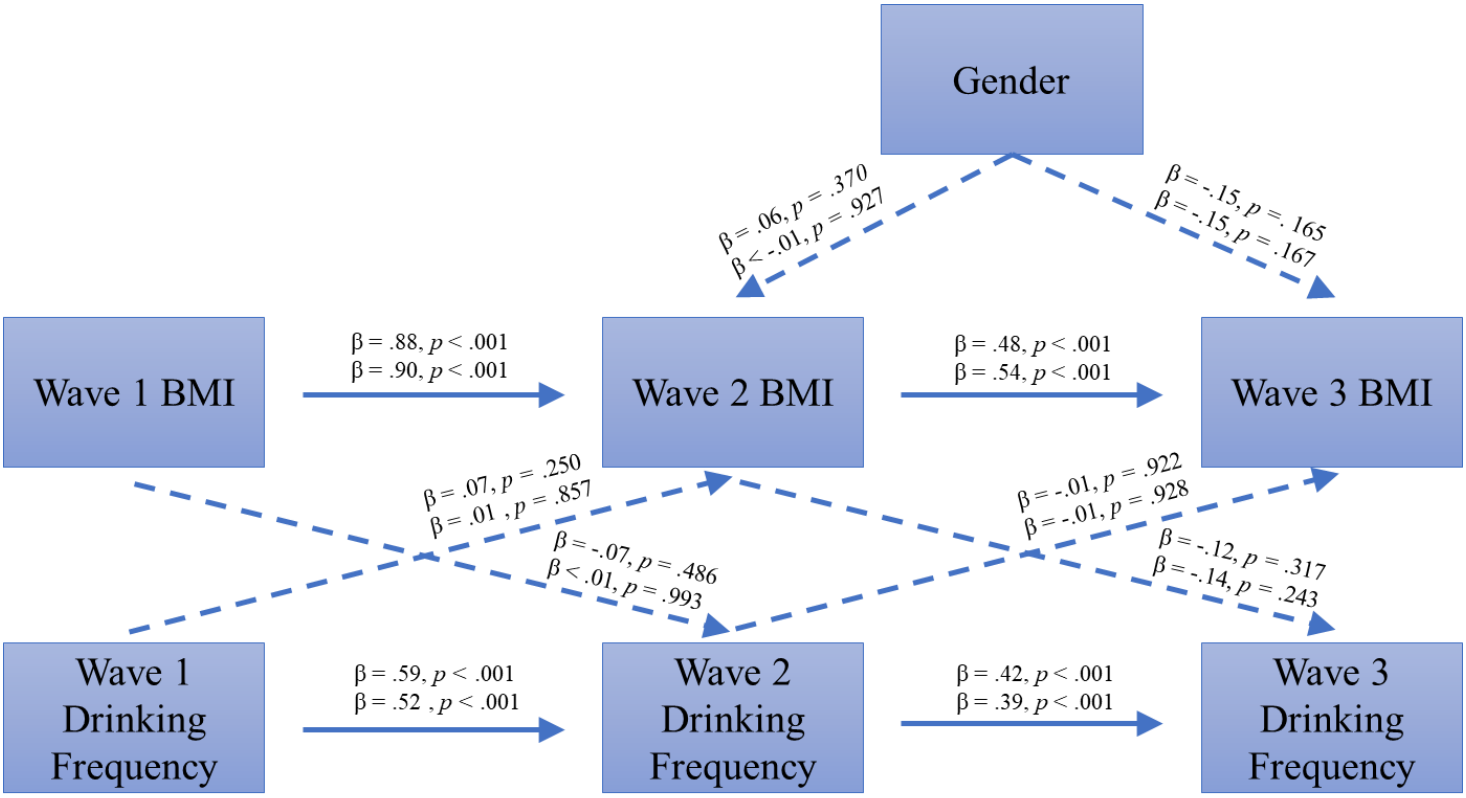

Figure 5.2. Model 1 - Associations between drinking frequency and BMI and gender and BMI, split by analysis type. Standardised regression coefficients are presented. Solid lines represent significant paths, dashed lines represent nonsignificant paths. Top line is complete case analysis, bottom line is with removal of participants who did not complete Wave 3.

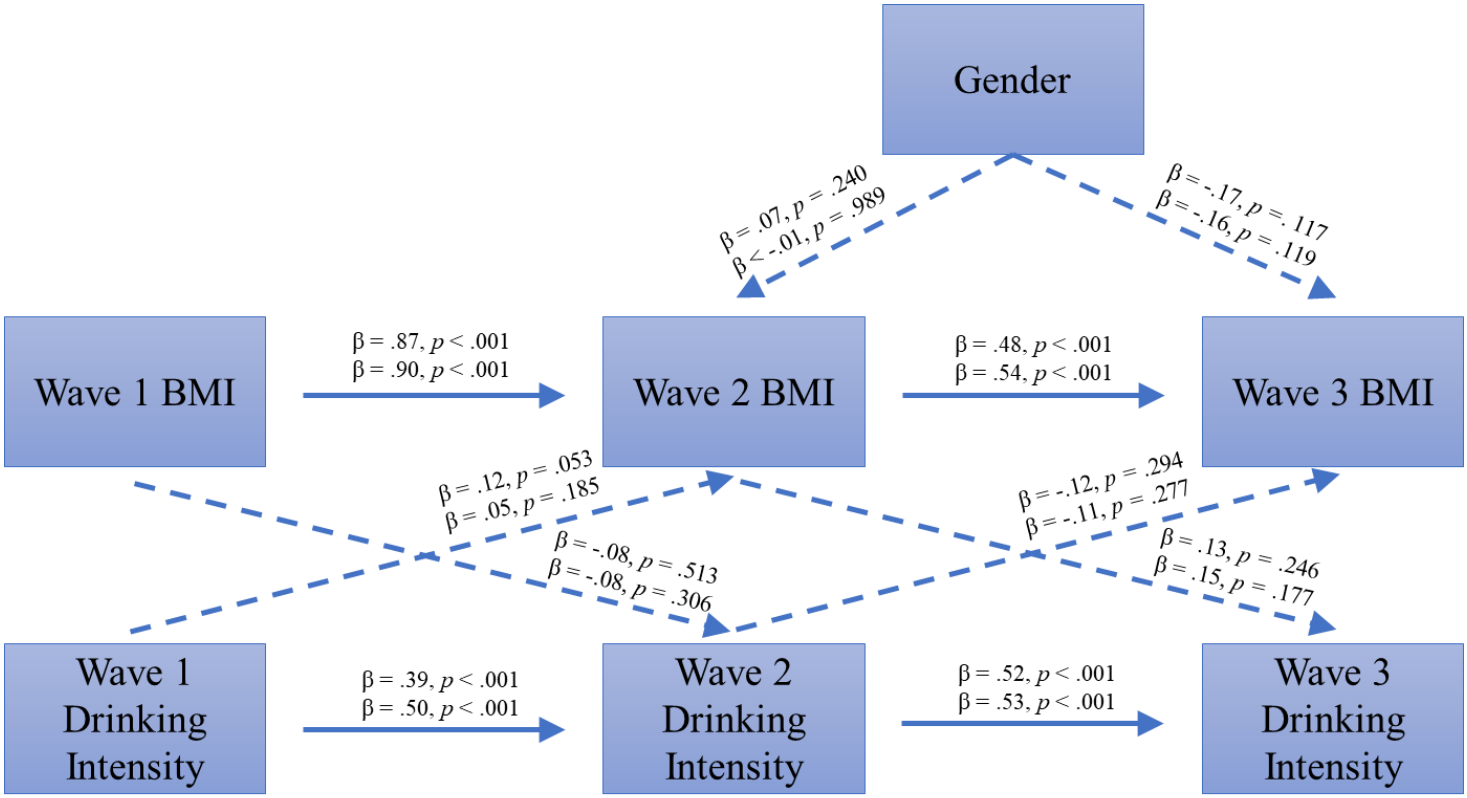

Figure 5.3. Model 2 - Associations between drinking intensity and BMI and gender and BMI, split by analysis type. Standardised regression coefficients are presented. Solid lines represent significant paths, dashed lines represent nonsignificant paths. Top line is complete case analysis, bottom line is with removal of participants who did not complete Wave 3.

5.5.3. Predictors of change in BMI (Table 5.3 and 5.4)

Findings from the regression analyses revealed that only Wave 1 BMI and change in CEBRACS scores (an increase in CEBRACS scores across time negatively correlated with BMI change across time) in model 3 and model 4 between Wave 1 and 2 significantly predicted change in BMI between Wave 1 and 2, whereas only change in CEBRACS scores significantly predicted change in BMI in model 3 and model 4 between Wave 1 and 3. The interaction term between motor impulsivity and change in drinking behaviour did not significantly predict change in BMI. See Tables 5.3 and 5.4 for a breakdown of each model used to predict changes in BMI.

Table 5.3. Model 3 - Hierarchical regression analysis showing change in IPAQ, change in CEBRACS, gender, baseline BMI, motor impulsivity, change in drinking frequency and the interaction term of motor impulsivity and change in drinking frequency as predictors of change in BMI between Wave 1 and 2 and Wave 1 and Wave 3.

	Wave 1 – Wave 2				Wave 1 – Wave 3			
Variable	Cumulative		Simultaneous		Cumulative		Simultaneous	
	R^2	F^a	β	p-value	R^2	F^a	β	p-value
Step 1	.14	10.27*			.14	10.31*		
Change in IPAQ			-.07	.569			.01	.923
Change in CEBRACS			-.17	.065			-.32	.037
Gender			-.03	.664			-.02	.661
Wave 1 BMI			-.25	.003			-.01	.958
Motor Impulsivity			.12	.165			-.12	.191
Change in Drinking Frequency			-.14	.787			.15	.308
Step 2	.15	9.20*			.15	9.22*		
Change in drinking Frequency x motor impulsivity			.11	.841			-.32	.355

* $p < .05$; a Step 1, df = (6,367); Step 2, df = (7,366)

Table 5.4. Model 4 - Hierarchical regression analysis showing change in IPAQ, change in CEBRACS, gender, baseline BMI, motor impulsivity, change in drinking intensity and the interaction term of motor impulsivity and change in drinking intensity as predictors of change in BMI between Wave 1 and 2 and Wave 1 and Wave 3.

	Wave 1 – Wave 2				Wave 1 – Wave 3			
Variable	Cumulative		Simultaneous		Cumulative		Simultaneous	
	R^2	F^a	β	p-value	R^2	F^a	β	p-value
Step 1	.15	10.69*			.15	10.42*		
Change in IPAQ			-.07	.530			.02	.865
Change in CEBRACS			-.17	.117			-.32	.023
Gender			-.03	.606			-.03	.581
Wave 1 BMI			-.26	.003			.01	.980
Motor impulsivity			.11	.203			-.10	.117
Change in Drinking Intensity			-.01	.972			.18	.495
Step 2	.15	9.25*			.15	10.61*		
Change in drinking intensity x motor impulsivity			-.01	.986			-.22	.439

* $p < .05$; a Step 1, df = (6,367); Step 2, df = (7,366)

5.5.4. Exploratory analyses

In order to investigate whether drinking behaviour is related to consumption of unhealthy foods, correlations were conducted to examine whether scores on the FFQ were related to drinking

frequency and drinking intensity. Findings revealed that Wave 1 FFQ scores were not significantly associated with Wave 1 drinking frequency (r = .058, p = .266) or with Wave 1 drinking intensity scores (r = .055, p = .287).

5.6. Discussion

The present study aimed to investigate whether alcohol consumption (measured separately as drinking frequency and drinking intensity) significantly predicted BMI over the course of 12 months in a first-year undergraduate sample. A further aim was to investigate whether motor impulsivity moderated the effect of drinking behaviour on BMI. Findings revealed that neither drinking frequency nor drinking intensity predicted changes in BMI at 6 months or 12 months. Furthermore, findings from the regression analysis showed that motor impulsivity and drinking behaviour did not interactively predict change in BMI. Therefore, both hypotheses were rejected.

The present findings add to the existing literature which have investigated whether alcohol consumption predicts weight change in undergraduate samples. Previous findings have been inconsistent as to whether drinking behaviour predicts weight-related outcomes in this group with some studies showing that drinking behaviour is associated with adiposity (Adams & Rini, 2007; Bodenlos et al., 2015; Deforche et al., 2015; de Vos et al., 2015; Economos et al., 2008; Lloyd-Richardson et al., 2008; Zagorsky & Smith, 2011) and others failing to show an association (Deliens et al., 2013; Fazzino et al., 2019; Kasparek et al., 2008; Pliner & Saunders, 2008; Pope et al., 2017).

One possible explanation for the previously inconsistent findings include the presence of confounding variables when examining the effect of drinking behaviour on weight. For example, compensatory behaviours in response to alcohol consumption have been shown to occur in undergraduates (Burke et al., 2010; Giles et al., 2009; Roosen & Mills, 2015). Findings in the present study showed that change in CEBRACS scores negatively predicted change in BMI – specifically when individuals increased in CEBRACS scores (indicating increased compensatory behaviours over time) from an earlier to a later time point, BMI decreased. Many of the previous studies have failed to control for potentially important confounding variables within the effect of drinking behaviour and weight change in first-year students. One study which did control for physical activity, found that

neither the number of weekly drinks nor the number of heavy drinking episodes per month predicted weight (Fazzino et al., 2019). However crucially, both Fazzino et al. (2019) and the present study had a much lower sample size than other studies which have shown a significant effect. Therefore, a lack of statistical power may explain the nonsignificant effect of drinking behaviour and its interaction term with motor impulsivity on BMI.

Findings also failed to show that change in drinking behaviour and motor impulsivity interacted to predict change in BMI. This result is in line with findings from Study 4 of Chapter 3 which found that motor impulsivity and change in food-related AB did not predict change in food intake between consumption of an alcoholic drink and a placebo-alcohol. Taken together, these findings suggest that those with higher levels of motor impulsivity are not more susceptible to alcohol-induced overeating and weight gain. As suggested in Chapter 3, this may be because alcohol-induced changes to state impulsivity could drive eating behaviour, rather than trait impulsivity. Therefore, trait impulsivity may not be an important determinant of alcohol-induced weight gain. Importantly, the present results are not consistent with Kase et al's. (2016) finding which showed that weight loss occurred after a reduction in alcohol consumption more so for highly impulsive individuals, possibly because when these individuals do drink, they are unable to inhibit prepotent responses. One explanation for this difference could be due to variation in how impulsivity was measured. The present study implemented a measure of trait impulsivity, whereas Kase et al. (2016) used a behavioural measure. Findings suggest that trait and behavioural measures of impulsivity are weakly correlated with each other (Cyders & Coskunpinar, 2011; Enticott et al., 2006; Reynolds et al., 2006a) and map onto different factor structures (MacKillop et al., 2016). Therefore, it may be the case that individual differences of inhibitory control, but not motor impulsivity, moderate the effect of alcohol-induced weight gain.

There are a number of limitations with the present study. Firstly, data collection at Wave 1 and 3 overlapped with the beginning of the semester. Many students engage in an orientation week ('freshers' week') during the first week of the semester. During this period, students may participate in several social events which may involve consumption of alcohol. Research has shown that drinking

behaviour during freshers' week is greater than a typical week (File et al., 1994). The reported drinking behaviour during these waves may have been heightened by atypical drinking behaviour during freshers' week, resulting in unrepresentative data. Additionally, improvements could be made regarding the measures used to capture drinking behaviour. In the present study, two items from the AUDIT-C were used. However, this measure provides limited variability as the responses are limited to 5 options for the drinking frequency question and 6 for the drinking intensity question. This measure was used because a previous study which categorised drinkers as low-risk and moderate-risk drinkers using the AUDIT was successful in detecting an effect of weight change (Lloyd-Richardson et al., 2008). However, instead, a measure which uses continuous data, such as the Timeline Follow back would be more sensitive to changes in drinking behaviour. Therefore, the combination of these methodological limitations may have contributed to a null finding. Future research should look to avoid collecting baseline data at a likely anomalous timepoint and instead ask participants to report their drinking behaviour prior to beginning University. Although this may result in greater inaccuracy as participants would need to recall further back, this would be a smaller cost than using extreme behaviour as baseline. The food frequency questionnaire included in this study, although adapted from a validated measure (Inchley et al., 2001), only captured a limited aspect of eating behaviour, focusing on unhealthy eating. This could have been improved by using alternative measurements, for example by using a validated scale to capture food frequency consumption of all food types (e.g., Brown & Ogden, 2004; Ogden et al., 2006) or by asking participants to complete a food diary at each wave of the survey. The present study did not measure the type of drinks typically consumed. However, types of alcohol can differ on its caloric content per standard drink. For example, beer which is high in carbohydrates, is more calorific than wine (Yeomans, 2010a). Different types of alcoholic drinks can also produce differing blood alcohol levels (Mitchell et al., 2014). Both of these factors may moderate any effect of alcohol consumption on BMI, and therefore the type of alcohol consumed should be included in future research. Lastly, current dieting status and dietary restraint was not measured in the present study. Evidence suggests that restrained eaters may alter their food intake in response to planned consumption of alcohol by having fewer eating episodes on days when they intend to consume alcohol (Luce et al., 2013). Therefore, although compensatory eating behaviours were

measured, future research may wish to further investigate whether dietary restraint moderates the association between drinking behaviour and adiposity.

In summary, the present study found that drinking behaviour did not predict changes in BMI in a sample of first year undergraduate students across a 12-month period. This suggests that drinking behaviour is not a determinant of increased BMI in this sample. Furthermore, findings revealed that trait motor impulsivity did not moderate the effect of drinking behaviour on change in BMI after controlling for confounding variables, suggesting that highly impulsive individuals are not more susceptible to alcohol-induced weight gain.

Chapter 6: General Discussion

6.1. Overview of thesis aims

Acute alcohol consumption has been shown to produce a caloric surplus relative to an alcohol-free drink (Kwok et al., 2019). However, many potential psychological mechanisms of this behaviour are either inconsistent or currently untested. The present thesis focused on identifying factors which may contribute to this effect. Specifically, mechanisms relating to cognitive control of eating behaviour and food reward within the context of acute alcohol consumption were examined. Furthermore, a dual-process model of appetite control was incorporated to examine whether alcohol-induced food intake and changes in BMI could be explained by an interaction of top-down and bottom-up processes.

The first aim of the thesis was to measure the influence of acute alcohol consumption on previously untested cognitive processes implicated in eating behaviour. Specifically, Chapter 2 investigated the role of alcohol-induced changes to episodic memories relating to food and how this affects subsequent food intake. To date, there has been no research which has examined whether acute alcohol consumption can alter recall of meal memories. The second aim of the thesis was to build on previous research and to measure the influence of acute alcohol consumption on both implicit and explicit measures of food reward. Chapter 3 investigated whether alcohol intoxication can affect self-report measures of food reward (i.e., appetite and snack urge ratings) as well as attentional bias towards food cues. Previous studies which have investigated the effect of acute alcohol consumption on food reward have provided mixed findings and suffer from methodological limitations, such as small sample sizes and heterogenous methodologies. The two studies in Chapter 3 addressed these inconsistent findings and methodological shortcomings. Additionally, Chapter 3 investigated whether the effect of food reward changed under two different dose - 0.3 g/kg (Study 3) and 0.6 g/kg (Study 4). The final aim of the thesis was to investigate whether a dual-process model of eating behaviour can account for alcohol-induced changes in acute eating behaviour and longer-term change in BMI. Study 4 (Chapter 3) investigated whether motor impulsivity and change in food-related attentional bias interactively predicted change in food intake between consumption of an alcoholic drink and a

placebo-alcohol. Chapter 4 examined whether motor impulsivity and changes in drinking behaviour interactively predicted change in weight in a cohort of first year Undergraduate students. Currently, the longitudinal relationship between drinking behaviour and BMI is mixed. The discrepancy in findings may in part be due to the different ways in which drinking behaviour is operationalised. Furthermore, compensatory behaviours which may offset an effect of drinking behaviour on BMI in previous studies, may have contributed to the previous mixed findings. Therefore, Chapter 4 measured the effect of drinking behaviour on BMI separately as drinking frequency and drinking intensity, and did so whilst controlling for potential confounding variables (i.e., compensatory behaviours).

6.2. Summary of findings

Chapter 2 presented findings from two studies which investigated how alcohol intoxication can affect recall of episodic memories relating to a recently consumed meal. In Study 1, participants consumed either an alcoholic drink (0.5 g/kg) or a placebo-alcohol drink, prior to consumption of a lunch meal. 30 minutes after the lunch meal, participants were presented with *ad libitum* access to cookies, after which participants were asked to recall details of the lunch meal. Findings revealed that alcohol consumption did not impair recall of the lunch meal relative to the placebo. However, *ad libitum* food intake did not differ between conditions. Furthermore, performance on the meal memory task did not mediate the effect of drink condition on *ad libitum* food intake. Study 2 explored whether acute alcohol consumption (0.6 g/kg) could impair or enhance recall of memories relating to a recently consumed meal, depending on whether alcohol was consumed before or after consumption of a meal and whether subsequent *ad libitum* food intake differed between these conditions. Findings revealed that memory for fullness was impaired when alcohol was consumed before a lunch meal, relative to an alcohol-free drink. However, consuming an alcoholic drink after a lunch meal did not improve meal memory recall, nor did it alter subsequent food intake.

Chapter 3 focused on how alcohol intoxication affects food reward. In Study 3, attentional bias towards food cues, self-report appetite, snack urge ratings and salivary response to foods and *ad libitum* food intake were measured to examine changes in food reward after consumption of an

alcoholic drink (0.3 g/kg) and a placebo-alcohol. Findings revealed that acute alcohol consumption did not produce changes in any measure of food reward relative to consumption of a placebo. Study 4 built directly upon these findings and investigated whether an alcohol-induced effect of food reward may be present after consumption of a greater dose of alcohol (0.6 g/kg). Food reward (as measured using food-related AB and self-report appetite and snack urge ratings) was compared between consumption of a 0.6 g/kg dose of alcohol and a placebo-alcohol. Additionally, alcohol-related AB and alcohol urge ratings were compared across drink conditions in order to provide a comparison of changes to reward between different types of appetitive stimuli. Findings revealed that consumption of the alcoholic drink did increase snack urge ratings, food-related AB, *ad libitum* food intake and alcohol urge ratings. This suggests that alcohol intoxication can affect food reward but only when alcohol is consumed above a certain level.

Study 4 of Chapter 3 also tested whether a dual-process model of eating behaviour could explain alcohol-induced changes in food intake. The interaction between trait motor impulsivity and change in food-related AB between drink conditions was included as a predictor variable of change in *ad libitum* food intake between drink conditions. Findings revealed that this interaction term was not associated with change in food intake, suggesting top-down and bottom-up processes do not interact to predict alcohol-induced changes in food intake. Chapter 4 investigated whether drinking behaviour (measured separately as how frequently someone drinks alcohol and the intensity of a drinking episode) can predict BMI change over the course of 12 months in a sample of first-year Undergraduate students. Findings revealed that neither measure of drinking behaviour predicted BMI across the 12-month period. Furthermore, from a dual-process perspective, the interaction between motor impulsivity and change in drinking behaviour (separately measured as drinking frequency and drinking intensity) revealed that neither measure of drinking behaviour and their interaction with motor impulsivity significantly predicted change in BMI. See Table 6.1 for a summary of study characteristics and findings from the four experimental studies included in the present thesis.

Table 6.1. Summary of study characteristics for the four experimental studies in the present thesis.

Study Number	Study design	Subjects (n) and sex	Age (years – mean and SD) and BMI (kg/m^2 – mean and SD)	Timing of beverage consumption	Test drink	Alcohol dose (g/kg)	Comparator	Test Food	Outcome of interest	Main findings
Study 1	Single-blind, between subjects randomised control trial. Participants consume either a placebo or alcoholic drink.	60 (30 females)	Age: 24.47 ± 10.13. BMI: 24.69 ± 4.70.	Consumed 10 minutes prior to a preload lunch meal and 60 minutes prior to *ad libitum* taste test	Vodka and diet lemonade	0.5 g/kg	Diet lemonade with vodka mist.	200 g chocolate chip cookies	Drink differences in recall of lunch items, appetite ratings and *ad libitum* food intake	Memory performance, appetite ratings and *ad libitum* food intake did not differ between conditions. Participants in the alcohol condition consumed more calories overall (test drink and *ad libitum* intake combined).
Study 2	Single-blind, between subjects randomised control trial. Participants consumed either a soft drink or alcoholic drink. The alcoholic drink was consumed either before or after a lunch meal.	72 (36 females)	Age: 24.31 ± 9.51. BMI: 24.57 ± 4.28.	Consumed test drink either 154 or 202 minutes before *ad libitum* taste test.	Vodka and diet lemonade	0.6 g/kg	Diet lemonade.	200 g chocolate chip cookies	Differences in recall of lunch meal, appetite ratings, and *ad libitum* food intake.	Memory of fullness was impaired when alcohol was consumed before a lunch relative to the soft drink. Total caloric intake was greater in the two alcohol conditions relative to placebo.

Table 6.1 Continued

Study Number	Study design	Subjects (n) and sex	Age (years – mean and SD) and BMI (kg/m^2 – mean and SD)	Timing of beverage consumption	Test drink	Alcohol dose (g/kg)	Comparator	Test Food	Outcome of interest	Main findings
Study 3	Single-blind within-subjects experiment.	44 (22 females)	Age: 25.55 ± 8.22. BMI: 25.98 ± 5.73	Consumed 25 minutes before *ad libitum* taste test.	Vodka and lemonade	0.3 g/kg	Diet lemonade with vodka mist.	200 g chocolate chip cookies, 200 g tortilla chips	Drink differences in food-related attentional bias, appetite ratings, snack urge ratings, *ad libitium* and total caloric intake.	No difference in attentional bias, appetite, snack urge ratings, *ad libitum* food intake. Total caloric intake was greater in the alcohol condition.
Study 4	Single-blind within-subjects experiment.	84 (71 females)	Age: 18.75 ± 1.13. BMI: 22.41 ± 3.54	Consumed 30 minutes before *ad libitum* taste test.	Vodka and lemonade	0.6 g/kg	Diet lemonade with vodka mist.	200 g chocolate chip cookies, 200 g tortilla chips	Drink differences in food and alcohol-related attentional bias, appetite ratings, snack urge ratings, *ad libitium* and total caloric intake.	Snack urge ratings, food-related AB, *ad libitum* and total caloric intake increased in the alcohol condition.

6.3. Theoretical implications

6.3.1. Alcohol's effect on cognitive processes

Findings from Chapter 2 provide evidence that acute alcohol consumption disrupts recall of memories relating to a meal, most likely due to disruptions during the encoding phase of memory formation. Previous research has implicated the importance of episodic memories relating to recently consumed food as a determinant of subsequent food intake (Higgs, 2016). Prior to this thesis, no studies had examined the effect of acute alcohol consumption on recall of a recently consumed meal. Study 1 tested this possibility and revealed that acute alcohol consumption did not impair recall of meal memory. Importantly, an inherent difficulty with testing for this effect is the extent to which the factor of meal memory on alcohol-induced eating can be measured in isolation, as alcohol-induced impairments to memory recall may correlate with the impairment of other cognitive factors implicated in alcohol-induced overeating (e.g., alcohol-induced disinhibition of eating behaviour). Therefore, Study 1 was not able to measure the isolated effect of memory impairment on food intake. Study 2 aimed to build on this and to separate apart the effect of meal memory recall on subsequent food intake from other factors. This was achieved by manipulating the order in which a lunch meal was presented relative to consumption of an alcoholic drink. Study 2 included two alcohol conditions which differed in terms of their level of alcohol intoxication at the point of encoding, but which both produced an elevated breath alcohol concentration at the point of recall and *ad libitum* food intake. This allowed for a more isolated observation of the effect of alcohol-induced changes to meal memory on subsequent food intake. Study 2 demonstrated that certain aspects of meal memory (i.e., memory for fullness) becomes impaired when an alcoholic drink is consumed prior to a lunch meal (compared with a soft drink). Study 2 also investigated whether the retrograde facilitation effect of memory occurs within the context of meal related memories. Results failed to show a difference in meal memory between these two conditions, suggesting that this attempted manipulation of memory recall was unsuccessful. Furthermore, general memory recall of did not differ between these two conditions, which goes against previous research (Knowles & Duka, 2004; Parker et al., 1980; Weafer et al., 2016). Therefore, the null findings and failure to replicate this effect may have been due to

methodological limitations, possibly relating to a small sample size and the timing of recall of the lunch meal memories, as the latter occurred whilst participants in the alcohol conditions were still intoxicated. Therefore, Study 2 is unable to conclude whether differences to meal memory (in the absence of other alcohol-induced determinants of eating behaviour) can alter changes to food intake. Taken together, findings Study 2 suggest that acute alcohol consumption does affect meal memory recall, however the extent to whether this contributes to alcohol-induced changes in eating behaviour remains unclear.

6.3.2. Food reward

Findings from Chapter 3 revealed that acute alcohol consumption affects aspects of food reward, but suggests that this effect may be dose dependent. This builds directly upon previous research which has investigated whether alcohol affects food reward in humans, with previously mixed findings. For example, self-reported hunger, food liking and desire to consume food have been shown to increase, decrease and show no change after an acute alcoholic drink, relative to an alcohol-free drink (Caton et al., 2005; Eiler et al., 2015; Hetherington et al., 2001; Poppitt et al., 1996; Rose et al., 2015; Schrieks et al., 2015; Westerp-Plantenga & Verwegen, 1999; Yeomans & Phillips, 2002). Similarly, alcohol's ability to alter food reward when captured using implicit measures of food reward have also been inconsistent (Adams & Wijk, 2020; Karyadi & Cyders, 2019; Monem & Fillmore, 2019). However, as mentioned in Section 1.3.1, evidence suggests that measures of food reward may become enhanced only when using higher levels of alcohol. For example, Rose et al. (2015) demonstrated that using a dose of 0.6 g/kg produced increased snack urge ratings compared with a placebo. Furthermore, Caton et al. (2004) showed that hunger ratings were significantly greater after consumption of 32 g of alcohol compared with 8 g and an alcohol-free preload. Building upon these findings, two studies in Chapter 3 tested this possibility and showed that snack urge ratings significantly increased after 0.6 g/kg, but not after 0.3 g/kg, relative to placebo. Another reason why an effect was found in the present thesis may have been due to a methodological strength of the present studies over previous research. Specifically, much of the previous research has used a fixed dose of alcohol (i.e., all participants consume the same amount of alcohol) when investigating the

effect of acute alcohol consumption on food reward. As bodyweight is an important determinant of the rate of absorption and blood alcohol content, providing a fixed dose to a sample of varying bodyweight will produce a wider range of blood alcohol content levels compared with when a dose is calculated according to bodyweight. Therefore, a previous failure to find an effect may have been because many participants failed to reach a blood alcohol level which is needed in order for alcohol to produce such an effect.

Interestingly, a dose-dependent response was not shown for appetite ratings. This may be because in Study 4, the test drink in both conditions consisted of a large volume of liquid. Therefore, feelings of fullness may have been high after ingestion of the test drink. An additional explanation for this finding is that participants were required to consume a light meal an hour before each test session. This may have meant that participants began the test session in a satiated state, which may have produced a suppressed effect of appetite ratings after consumption of the test drink. Whereas, other studies which have shown an effect on hunger ratings (e.g., Caton et al., 2004) implemented a longer period of fasting prior to alcohol consumption. Therefore, an effect on appetite ratings may have been found if participants were asked to fast for an extended period of time prior to alcohol consumption. A fasting period was not implemented in the current studies due to ethical considerations regarding the increased risk of an adverse reaction to alcohol consumption when consumed without recent consumption of a meal.

Attentional bias was also investigated in this thesis. Previous research has suggested that acute alcohol consumption does not increase attentional bias towards certain foods. For example, Adams and Wijk (2020) found that the magnitude of attentional bias towards high-energy vs low-energy food cues did not differ between consumption of a 0.4 g/kg dose of alcohol and a placebo-alcohol. Furthermore, Monem and Fillmore (2019) failed to show a dose-dependent response on the magnitude of food-related attentional biases after consumption of a placebo-alcohol, 0.3 g/kg or 0.65 g/kg dose of alcohol. One explanation for these previous null findings may be due to the sample size used. In both of the previously mentioned studies, the number of participants in each condition did not exceed 23, meaning that these studies were powered to detect only a medium-to-large effect. Recent

research by Hardman et al. (2020) has demonstrated that food-related AB is only weakly associated with motivational states (i.e., snack urge ratings and appetite). Therefore, a failure to observe an effect of drink on food-related AB may have been due to a lack of statistical power. In support of this suggestion, Study 4 (which was powered to detect a small effect size between drink conditions) found an effect on food-related AB after consumption of a 0.6 g/kg dose of alcohol, relative to placebo, but not in Study 1 after a dose of 0.3 g/kg.

An additional finding from Studies 3 and 4 was the lack of correspondence between attentional bias and motivational state. In both studies, food-related attentional bias did not correlate with snack urge ratings. In Study 4, alcohol-related attentional bias did not correlate with alcohol urge ratings. These null findings do not support theories which argue that attentional bias is driven by motivational states (e.g., Field et al., 2016). Although neither studies were powered to detect an effect size reported by Hardman et al. (2020), caution should be made when using attentional bias as a measure of motivational state.

Collectively, acute alcohol consumption appears to enhance food reward after a moderate but not low dose of alcohol. However, findings from the present studies suggest that this does not include enhanced appetite, but rather an enhancement of snack urge ratings as well as implicit measures such as attentional bias. Crucially though, findings from Study 4 suggest that alcohol-induced change in attentional bias does not predict alcohol-induced change in food intake, therefore the current thesis did not find evidence to suggest that food reward directly predicts food intake in this context.

6.3.3. Dual-process effect of alcohol on food intake

Chapters 3 and 4 explored whether alcohol-induced increases in food intake occur through an interaction of top-down and bottom-up processes. Specifically, studies 4 and 5 examined whether trait motor impulsivity moderates the effect of alcohol-induced changes to food intake and BMI, respectively. Findings from Study 4 revealed that an interaction of trait motor impulsivity and change in food-related AB did not significantly predict change in food intake. Study 5 predicted a dual-process account of drinking behaviour in the context of longer-term weight change. However, findings revealed that trait motor impulsivity did not interact with change in either measure of

drinking behaviour (drinking frequency or drinking intensity) to predict change in BMI, again suggesting that individuals who have difficulty to inhibit bottom-up processes (i.e., individuals with increased levels of impulsivity) are not more susceptible to alcohol-induced changes to BMI.

Previous research has shown that the interaction of top-down and bottom-up processes significantly predict food intake and BMI (Appelhans et al., 2011; Bickel et al., 2014; Davis et al., 2010; Epstein et al., 2014; Jarmolowicz et al., 2014; Nederkoorn et al. 2006; Weller et al., 2008). One reason why both Study 4 and 5 did not provide support for this interaction effect may have been due to measuring trait rather than state impulsivity. Given that acute alcohol consumption increases state impulsivity (Abroms et al., 2003; Christiansen et al., 2016; de Wit et al., 2000; Fillmore & Vogel-Sprott, 2000; Marczinski & Fillmore, 2003; Marinkovic et al., 2012), alcohol consumption may have produced an increase in impulsive behaviours across all participants, therefore reducing the importance of baseline trait impulsivity as a predictor of alcohol-induced changes in food intake. A failure to show an interaction effect may have been due to alcohol's ability to increase state impulsivity which may then have reduced the predictive power of trait impulsivity. Furthermore, Kase et al. (2016) demonstrated that individual differences in impulsivity moderated the effectiveness of alcohol reduction on weight loss, when measured using behavioural impulsivity (i.e., performance on a Go/No-Go Task). It is possible that certain types of impulsivity are more important in alcohol-induced overeating than others. In support of this, research has shown that self-report and behavioural measures of impulsivity are weakly related with each other (Cyders & Coskunpinar, 2011; Enticott et al., 2006; Lijffijt et al., 2004; Reynolds et al., 2006a). Studies which have aimed to identify the factor structure of impulsivity suggest that trait impulsivity is separate to behavioural measures (MacKillop et al., 2016) and that different behavioural measures represent different components (Reynolds et al., 2006a). Therefore, it is possible that measures of behavioural impulsivity (particularly those which measure inhibitory control) better capture behaviours which are important in determining alcohol-induced overeating.

6.3.4. Caloric Intake

Studies across Chapter 3 and 4 investigated whether acute alcohol consumption can affect food intake. In Chapter 4, much like the findings for food reward, food intake did increase after a dose of 0.6 g/kg, but not after a dose of 0.3 g/kg. In Chapter 3, neither of the two studies showed an increase in food intake after consumption of an alcoholic drink relative to a placebo or soft drink. In the case of Study 2, a failure to show a significant difference between consumption of alcohol and a soft drink may have resulted from a small sample size, but also from the extended delay between consumption of the test drink and *ad libitum* taste test, which has previously been suggested as a factor which reduces the effect of acute alcohol consumption on food intake (Yeomans, 2010a). As participants' blood alcohol contents would be on the descending limb of the blood alcohol curve, the pharmacological effects of alcohol on food intake were likely to be minimal despite ingesting a dose of 0.6 g/kg.

In Study 1, one reason for failing to find a difference between conditions may have been due to a lack of statistical power. This study was powered to detect a medium-to-large effect between conditions as research has found a similar effect size using a similar dose (Christiansen et al., 2016; d = 0.61). However, the effect size from the nonsignificant difference in Study 1 was small-to-medium (d = 0.34). This effect size may have been smaller because a slightly weaker dose was used (0.5 g/kg, whereas Christiansen et al. used 0.6 g/kg) but also because participants consumed a lunch meal, followed by a 30-minute break before completing the *ad libitum* taste test. As consumption of food increases the elimination rate of alcohol (Ramchandani et al., 2001), the blood alcohol content of participants would likely have been lower at the point of *ad libitum* food intake than those in Christiansen et al's (2016) study, who did not consume a preload meal. An alternative possibility is that the effect size in the study by Christiansen et al. (2016) is inflated due to a small sample size. This is plausible because a methodologically similar study (in terms of the dose and type of alcohol used) failed to show a significant effect of alcohol (0.6 g/kg) on food intake (Rose et al., 2015). Furthermore, Study 4 which used a dose of 0.6 g/kg, found a small-to-medium effect size (d = 0.36). This study has, to date, the greatest statistical power to observe a difference in food intake between an

alcoholic drink and an alcohol-free placebo. Therefore, this effect size is likely to be a better estimate of the difference in food intake using the same dose and type of alcoholic drink, than previous studies which have implemented a smaller sample size. Studies which are not powered to detect a small-to-medium effect size may therefore lack statistical power to observe a significant difference.

A dose-dependent effect of alcohol-induced food intake is inconsistent with Kwok et al. (2019), who found that a low dose (< 30 g or < 0.6 g/kg) but not a high dose of alcohol (≥ 30 g or ≥ 0.6 g/kg) produces greater food intake relative to an alcohol-free control drink. However, the analysis by Kwok et al. (2019) pooled together heterogenous data which varied on the type of alcohol and dose used (including both fixed and weight-dependent doses), therefore potentially reducing the effect. Instead, findings from Chapter 4 provide good evidence for a dose-dependent effect when using a weight-dependent dose with a sufficient sample size, and when consuming an alcoholic spirit. This latter point is an important distinction because consumption of spirits produces a greater blood alcohol content compared with wine or beer at the same dose (Mitchell et al., 2014), Therefore, studies which use other types of alcohol at the same dose may not achieve the same blood alcohol content, potentially producing different patterns in eating behaviour.

Findings regarding total caloric content are consistent with the wider literature (Kwok et al., 2019). All four studies across Chapters 3 and 4 demonstrated that, relative to an alcohol-free drink, consumption of an alcoholic drink produced greater total caloric intake suggesting that the additional calories consumed from alcohol are not immediately compensated for in a subsequent eating episode. This is likely to be in part because energy consumed in liquid form is poorly compensated for, relative to semi-solid and solid foods (Almiron-Roig et al., 2013). Present findings suggest that this effect occurs across a range of doses and even after an extended period (≥ 2.5 hours, as implemented in Study 2).

Lastly, findings from Chapter 3 found that the nonsignificant difference in food intake between drink conditions was consistent when performing the analysis separately for males and females. This is in line with previous research suggesting that an alcohol-induced effect on food intake is consistent in both males and females (Kwok et al., 2019), despite sex differences in how

alcohol is metabolised (Ammon et al., 1996; Thomasson et al., 1995). Furthermore, Chapter 3 found no evidence of sex differences in alcohol-induced changes to meal memory performance, suggesting that acute alcohol consumption may impair meal memory similarly in males and females.

6.4. Practical Implications

Findings from the present thesis add to growing evidence that acute alcohol consumption does produce a caloric surplus. This has important practical implications, as it implicates reductions in alcohol consumption as a potentially beneficial weight-loss strategy. Further, findings from Chapter 4 suggest that even small doses of alcohol can produce a greater total caloric intake, relative to consumption of an alcohol-free drink. However, Study 4 showed that greater doses of alcohol appear to increase food intake in addition to the calories consumed from alcohol, suggesting that greater doses of alcohol may make individuals more susceptible to weight gain. Although Study 5 failed to show that drinking intensity predicts change in BMI, this suggestion is in line with previous research implicating the number of alcohol units consumed during a drinking episodic as a predictor of weight gain (Breslow & Smothers, 2005; French et al., 2010; Tolstrup et al., 2005). Therefore, a practical use of these findings would be to advise consumers of alcohol who wish to reduce the risk of weight gain, that they should avoid consuming large amounts of alcohol in a single drinking episode.

Findings also further implicate enhanced food reward as a factor by which acute alcohol consumption increases food intake. For individuals who live in an obesogenic environment (characterised in part by ubiquitous availability of calorie-dense foods), resisting consumption of unhealthy foods as the result of increases in food reward after acute alcohol consumption may be particularly difficult.

6.5. Strengths and Limitations

There are several strengths of the studies included in this thesis. Firstly, in all of the experimental studies, alcohol dose was based on bodyweight. As previously mentioned, this type of dose provides a more consistent blood alcohol content across participants relative to a fixed dose. Secondly, in three of the four experimental studies, an equal number of males and females were

recruited in order to produce a more representative sample. Power calculations were used to determine the sample size for each experimental study, meaning that these studies were sufficiently powered to detect a likely effect. The methodology and analysis plan of two of the experimental studies were also pre-registered, meaning that the analyses were decided upon before data collection began. Cover stories were implemented across all experimental studies to help minimise aim guessing, as awareness of the true experimental aims can affect behaviour (Kersbergen et al., 2019). In studies 1, 3 and 4, a placebo was implemented in order to control for anticipated effects of alcohol on eating behaviour. Study 4 demonstrated that participants believed that the number of alcohol units in the placebo drink was significantly greater than zero, indicating that participants believed there to be alcohol in this condition.

There were some limitations throughout the thesis. Firstly, all of the laboratory experiments were conducted in a typical laboratory environment. This may be problematic if participants are aware that food intake is being measured, as several studies have shown that food intake is suppressed when people are made to feel aware that their food intake is being measured (Robinson et al., 2015; Robinson et al., 2014a; Robinson et al., 2016). Furthermore, evidence suggests that the effect of an experimental manipulation predicted to alter food intake is greater in a real-world setting compared with in a laboratory (Gough et al., 2021). Therefore, one possibility is that in a real-world environment, the effect of alcohol consumption on eating behaviour may differ when conducted in the laboratory. Similarly, alcohol-related environments (i.e., a laboratory with the appearance of a bar) has been shown to increase short-term alcohol consumption (Lau-Barraco & Dunn, 2009; Moss et al., 2015), possibly due to contextual cues initiating alcohol-related behaviours (Moss & Albery, 2009). Although Rose et al. (2015) found no difference in food intake between a standard laboratory and a semi-naturalistic bar laboratory, it is possible that a fully naturalistic environment may produce greater levels of alcohol-induced food intake.

Throughout the experiments, palatability of the test drink and pre-load lunch meals (where appropriate) was not assessed. Importantly, perceived palatability of the beverage and preloads may have affected subsequent food intake, as some evidence suggests that eating of a preferred food can

produce greater subsequent desire-to-eat and hunger ratings (Hill et al., 1984). However, findings suggest that acute alcohol consumption alone does not increase pleasantness of certain foods (Caton et al., 2005). Nevertheless, if participants found one test drink less palatable than another, this may have affected subsequent food intake. Therefore, future research should ensure that palatability ratings of the test drink and any preload meals are recorded.

Another important limitation of the thesis was the limited range of doses used, with a maximum dose of 0.6 g/kg. This maximum dose fails to fully examine how greater levels of drinking behaviour (i.e., binge drinking) can affect determinants of eating behaviour. Although loosely defined, binge drinking by the NHS is considered as drinking more than six and eight units of alcohol in a single session for women and men, respectively. Given that binge drinking and heavy drinking are implicated in weight gain (Traversy & Chaput, 2015), binge drinking may produce a greater effect on food intake compared with smaller doses. However, administering doses similar to that of binge drinking would be unethical in a laboratory setting. One way in which future research could investigate this is through observing behaviours in the real-world.

Lastly, none of the experimental studies examined food consumption for an extended period after alcohol consumption. Although previous research has investigated the effect of alcohol consumption across several hours (Caton et al., 2004), the present studies could have benefitted from observing food intake across a longer period of time (e.g., 24 hours) in order to see whether caloric compensation (through decreased energy intake or increased expenditure) occurs after consumption of alcohol. This suggestion applies to investigations of acute alcohol consumption on caloric balance in general.

6.6. Future Research

The current findings provide a significant contribution towards the topic of alcohol consumption on food intake and BMI. However, there are clear avenues for subsequent research. For example, future research should further investigate whether alcohol intoxication can increase food intake through an interaction of top-down and bottom-up processes. Although this was explored in the current thesis, the studies did not examine state changes in top-down processes (i.e., impulsivity). As

acute alcohol consumption decreases inhibitory control (Abroms et al., 2003; Christiansen et al., 2016; de Wit et al., 2000; Fillmore & Vogel-Sprott, 2000; Marczinski & Fillmore, 2003; Marinkovic et al., 2012), it is possible that alcohol-induced changes to state impulsivity, rather than trait impulsivity, interact with alcohol-induced changes to motivational orientation of food, resulting in greater food intake.

Future research should also elucidate whether alcohol-induced changes to food intake and determinants of eating behaviour do increase in a dose-dependent manner. Findings from Chapter 3 suggest that this pattern does exist, as effects on food reward and intake were seen after consumption of 0.6 g/kg, but not after 0.3 g/kg. However, this was examined across two studies. Stronger evidence for a dose-dependent case will come from investigations which measure varying levels of alcohol consumption within the same sample.

6.7. Concluding remarks

This thesis has investigated the role of psychological mechanisms which may contribute towards alcohol-induced changes in food intake and BMI. The aims of this thesis were to see how cognitive and reward-related components could affect these outcomes. Overall, findings revealed that acute alcohol consumption alters behaviours implicated in determining food intake. Specifically, findings showed that recall of memories related to a recently consumed meal were impaired after consumption of alcohol, however this did not predict food intake. Findings also provided further clarity regarding whether acute alcohol consumption increases food reward, whereby a moderate (but not low) dose of alcohol was shown to increase measures of food reward and food intake. However, the thesis failed to provide support for a dual-process account of alcohol-induced changes in food intake. Similarly, trait motor impulsivity did not moderate the effect of drinking behaviour on BMI, questioning the predictive power of this model within the context of drinking behaviour. Taken together, acute alcohol consumption does appear to increase food intake at a moderate dose. However, the importance of the measured cognitive and reward processes within this thesis remains unproven and requires further investigation.

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Chapter 8: Appendices

8.1. Appendix A: Supplementary results (Chapter 2)

Study 1

Snack Urge Ratings

A 2 (drink; placebo, alcohol drink) x 4 (time; baseline, post-drink, post-lunch, pre-taste test) mixed ANOVA with drink as a between-subjects factor and time as a within-subjects factor revealed a significant main effect of time $F(3, 174) = 5.61$, $p = .001$, $\eta_p^2 = .088$. Bonferroni corrected pairwise comparisons revealed that ratings were significantly higher at post-drink compared with post-meal ($p = .012$; mean difference = 37.25; 95% CI [5.86, 68.64]) and pre-taste test ratings ($p = .037$; mean difference = 27.57; 95% CI [-54.07, -1.04]). There was also a significant main effect of drink type $F(1,58) = 6.88$, $p = .011$ $\eta_p^2 = .11$ with significantly greater snack urge ratings after consumption of the alcoholic drink compared with the placebo. Lastly, there was a nonsignificant interaction between drink type and time $F(3, 174) = 1.16$, $p = .328$, $\eta_p^2 = .02$.

BAES

Two separate 2 (drink; placebo, alcohol drink) x 4 (time; baseline, post-drink, post-lunch, post-taste test) mixed ANOVAs were conducted with drink as a between-subjects factor and time as a within-subjects factor, one for scores on the sedation scale, one for total scores on the stimulation scale. Mauchly's test indicated that the assumption of sphericity had been violated for the main effect of time $\chi^2(5) = 30.02$, $p < .001$, Greenhouse-Geisser corrected tests are reported ($\varepsilon = .718$) For the sedation scale, there was a significant main effect of time $F(2.12, 122.84) = 6.47$, $p < .001$, $\eta_p^2 = .10$. Bonferroni corrected pairwise comparisons revealed that sedation scores were significantly lower at baseline compared with post-drink ($p = .016$; mean difference = 3.67; 95% CI [-6.85, -.49]), post-lunch ($p = .040$; mean difference = 4.74; 95% CI [-9.35, -.14]) and post-taste test ($p < .001$; mean difference = 4.76; 95% CI [-7.61, -1.91]). No other significant difference between time points were found ($p > .05$). There was a nonsignificant main effect of condition $F(1,57) = .01$, $p = .910$, $\eta_p^2 < .01$. There was also a nonsignificant drink x time interaction $F(2.16, 122.84) = .12$, $p = .899$, $\eta_p^2 < .01$.

For the stimulation scale, there was a significant main effect of time $F(3, 171) = 10.28$, $p < .001$, $\eta_p^2 = .15$. Bonferroni corrected pairwise comparisons revealed that stimulation scores were significantly lower at baseline compared with post-drink scores ($p = .039$; mean difference = 2.81; 95% CI [-5.52, -0.10]) and significantly greater than post-taste test scores ($p = .010$; mean difference = 2.96; 95% CI [0.51, 5.41]). Post-taste test scores were significantly lower than both post-drink ($p < .001$; mean difference = 5.77; 95% CI [2.65, 8.89]) and post-lunch scores ($p = .003$; mean difference = 4.42; 95% CI [1.13, 7.71]). There were no other significant differences ($p > .05$). There was also a significant drink x condition interaction $F(3, 171) = 4.66$, $p = .004$, $\eta_p^2 = .08$. This was the result of a nonsignificant difference between conditions at baseline $t(58) = .39$, $p = .700$, $d = 0.10$, post-drink $t(57) = -1.31$, $p = .196$, $d = 0.34$ and post-taste test $t(58) = -.89$, $p = .380$, $d = 0.23$, but a significant difference at post-lunch $t(58) = -2.55$, $p = .013$, $d = 0.66$, with higher scores in the placebo condition. There was a nonsignificant main effect of condition on stimulation scores $F(1,57) = 1.71$, $p = .196$, $\eta_p^2 = .03$

Descriptive statistics of appetite ratings, snack urge ratings and BAES.

Table A1. Mean (± standard deviation) appetite, snack urge, BAES stimulation, BAES sedation at each time point, split by drink condition.

	Placebo	Alcohol Drink
Appetite Ratings (Baseline)	74.28 (± 37.46)	84.07 (± 42.39)
Appetite Ratings (Post-drink)	81.59 (± 42.63)[1]	100.47 (± 40.93)
Appetite Ratings (Post-lunch)	23.52 (± 21.40)	32.13 (± 26.72)
Appetite Ratings (Pre-taste test)	37.45 (± 25.03)	41.93 (± 22.02)
Snack Urge Ratings (Baseline)	175.34 (± 64.22)	204.70 (± 82.48)
Snack Urge Ratings (Post-drink)	186.28 (± 79.45)[1]	232.83 (± 91.82)
Snack Urge Ratings (Post-lunch)	139.76 (± 76.94)	197.67 (± 108.65)
Snack Urge Ratings (Pre-taste test)	148.62 (± 79.03)	208.43 (± 91.97)
BAES Stimulation (Baseline)	34.52 (± 8.50)	33.37 (± 10.79)
BAES Stimulation (Post-drink)	34.79 (± 9.45)[1]	38.70 (± 13.12)
BAES Stimulation (Post-lunch)	31.97 (± 8.22)	38.83 (± 12.04)
BAES Stimulation (Post-taste test)	29.72 (± 7.99)	32.23 (± 10.71)
BAES Sedation (Baseline)	16.97 (± 10.31)	16.17 (± 11.51)
BAES Sedation (Post-drink)	19.93 (± 9.16)[1]	20.53 (± 12.78)
BAES Sedation (Post-lunch)	21.59 (± 11.43)	21.03 (± 15.36)
BAES Sedation (Post-taste test)	21.55 (± 10.98)	21.10 (± 11.39)

Note. [1] = data missing from one participant.

BrAc readings

Table A2. Mean (± standard deviation) breath alcohol concentration (mg/L) split by condition and time point.

	Baseline	Post-drink	Post-break	Post-taste test
Alcoholic Drink	0 (0)	0.22 (0.09)	0.18 (0.59)	0.15 (0.05)
Placebo	0 (0)	0 (0)	0 (0)	0 (0)

Study 2

Snack Urge Ratings

A 3 (drink; soft drink, pre-meal drink, post-meal drink) x 3 (time; baseline, post-lunch, post-break) mixed ANOVA was conducted with drink as a between-subjects factor and time as a within-subjects. This revealed a main effect of time $F(2,136) = 16.58$, $p < .001$, $\eta_p^2 = .196$. Bonferroni corrected pairwise comparisons revealed that snack urge ratings were significantly greater at baseline than post-lunch ($p = .021$; mean difference = 25.26; 95% CI [3.02, 47.50]) and significantly lower at baseline than post-break ($p = .011$; mean difference = 27.15; 95% CI [-49.20, -5.11]). Post-lunch ratings were significantly lower than post-break ratings ($p < .001$; mean difference = 52.41; 95% CI [-75.15, -29.67]). There was also a main effect of drink $F(2,68) = 3.26$, $p = .045$, $\eta_p^2 = .09$. Bonferroni corrected comparisons revealed however that no drink conditions significantly differed from one another: soft drink and pre-meal drink ($p = .052$; mean difference = 37.33; 95% CI [-74.94, .28]); soft drink and post-meal drink ($p = .200$; mean difference = 28.25; 95% CI [-65.46, 8.96]); pre-meal and post-meal ($p = 1.00$; mean difference = 9.08; 95% CI [-28.533, 46.69]). Lastly, there was a significant drink x time interaction effect $F(4,136) = 3.95$, $p = .005$, $\eta_p^2 = .10$. Univariate ANOVAs revealed that this interaction effect was due to a nonsignificant difference between drink conditions at baseline $F(2, 69) = .67$, $p = .514$, $\eta_p^2 = .02$ and at post-break $F(2, 69) = .55$, $p = .582$, $\eta_p^2 = .02$, but a significant difference at post-lunch $F(2,68) = 8.73$, $p < .001$, $\eta_p^2 = .20$. Bonferroni corrected comparisons revealed that those in the soft drink condition had significantly lower snack urge ratings at post-lunch compared with the pre-meal drink condition ($p < .001$; mean difference = 87.11; 95% CI [-138.30, -35.93]).

Descriptive statistics of appetite ratings and snack urge ratings.

Table A3. Mean ± (standard deviation) appetite ratings and snack urge ratings for each time point, split by drink condition).

	Soft Drink	Pre-meal Drink	Post-meal Drink
Appetite Ratings (Baseline)	111.50 (± 38.01)	127.70 (± 36.57)	105.92 (± 35.27)
Appetite Ratings (Post-lunch)	36.13 (± 27.59)	79.04 (± 52.64)	45.38 (± 35.27)
Appetite Ratings (Post-break)	101.38 (± 38.01)	114.22 (± 47.32)	109.96 (± 45.15)
Snack Urge Ratings (Baseline)	195.13 (± 67.09)	202.39 (± 67.61)	215.71 (± 54.52)
Snack Urge Ratings (Post-lunch)	135.58 (± 61.11)	222.70 (± 92.44)[1]	179.17 (± 56.47)
Snack Urge Ratings (Post-break)	218.83 (± 69.78)	236.43 (± 61.50)	239.42 (± 80.08)

Note. [1] = data missing from one participant.

BrAc readings

Table A4. Mean (± standard deviation) breath alcohol concentration (mg/L) split by condition and time point.

	Baseline	Post-drink	Post-break
Pre-meal drink	0 (0)	0.29 (0.12)[1]	0.12 (0.04)
Post-meal drink	0 (0)	0.23 (0.10)	0.17 (0.05)
Soft Drink	0 (0)	0 (0)	0 (0)

Note. [1] = data missing from one participant.

8.2. Appendix B: Supplementary results (Chapter 3)

Study 3

Gaze Dwell Times

Table 1 shows estimates for internal reliability across trial type and condition. Estimates of internal reliability revealed that overall internal reliability for mean bias scores on palatable with control trials, and internal reliability for all combined trials in the alcohol condition and also in the placebo condition, and both combined, were above the acceptable criteria of .7 (Kline, 1999).

Table A5. Internal reliability of AB scores, split by trial type and condition type (values are McDonald's Omega).

	Palatable and Control	Unpalatable and Control	Palatable and Unpalatable
Alcohol	.76	.61	.79
Placebo	.82	.74	.69
Total	.81	.74	.70

Breath alcohol levels

Table A6. Breath alcohol concentration (mg/L) scores (mean ± SD) at each time point, split by drink condition

	Baseline	Post-drink	Post-taste test
Alcoholic Drink	0 ± (0)	.14 ± (.06)	.12 ± (.05)
Placebo	0 ± (0)	0 ± (0)	0 ± (0)

Eye-tracking data (Gaze Direction Bias)

Gaze direction bias towards any image type was shown to not significantly differ between conditions for palatable and unpalatable trials $t(36) = -.484$, $p = .632$, unpalatable and control trials $t(36) = .069$, $p = .946$, and palatable and unpalatable trials $t(36) = .722$, $p = .47$ (see table 3)

Visual Probe Task (Reaction time data)

Reaction time data were subject to a trimming procedure (see Schoenmakers et al., 2008). Reaction times faster than 200 ms, slower than 2000 ms and then three standard deviations above the individual mean were removed prior to analysis. This led to the removal of 1.55% of trials from the visual probe task. Reaction time data were lost for one participant due to error in recording the data.

Three 2x2 repeated measures ANOVAs with condition (alcohol, placebo) and probe position (congruent, incongruent) were conducted separately on the three different image pair types. This revealed three nonsignificant interactions of condition and probe position for the comparison between palatable and control images $F(1, 42) = 0.03$, $p = .874$, $\eta_p^2 = .001$, palatable and unpalatable images $F(1, 42) = 2.39$, $p = .130$, $\eta_p^2 = .054$, and unpalatable with control images $F(1, 42) = 3.28$, $p = .077$, $\eta_p^2 = .073$. The ANOVAs also revealed three nonsignificant main effects of condition for the comparison between palatable and control images $F(1, 42) = 0.04$, $p = .845$, $\eta_p^2 = .001$, palatable and unpalatable images $F(1,42) = 0.76$, $p = .388$, $\eta_p^2 = .018$, and unpalatable and control $F(1,42) = 0.98$, $p = .327$, $\eta_p^2 = .023$. Finally, the ANOVAs also revealed three nonsignificant main effects of probe position for the comparison between palatable and control images $F(1,42) = 0.11$, $p = .748$, $\eta_p^2 = .002$, palatable and unpalatable images $F(1,42) = 0.17$, $p = .684$, $\eta_p^2 = .004$, and unpalatable and control images $F(1,42) = 0.11$, $p = .741$, $\eta_p^2 = .003$.

Study 4

Modified Stroop Task

Table A7. Internal reliability of AB scores, split by AB task and condition type (values are McDonald's omega).

	Food pairs	Alcohol pairs
Alcoholic Drink	.62	.83
Placebo	.51	.60
Total	.57	.68

Table A8. Breath alcohol concentration (mg/L) scores (mean ± SD) at each time point, split by drink condition

	Baseline	Post-drink	Post-taste test
Alcoholic Drink	0 ± (0)	.20 ± (.06)	.20 ± (.10)
Placebo	0 ± (0)	0 ± (0)	0 ± (0)

Subjective Intoxication scores

A 2 (drink; alcohol, placebo) x 2 (baseline, post-drink) repeated measures ANOVA was conducted on each measure of the subjective intoxication scale, these were: lightheaded, irritable, stimulated, alert, relaxed and contented. Firstly, there was a significant main effect of drink on lightheaded scores $F(1,83) = 119.46$, $p < .001$, $\eta_p^2 = .59$, with those in the alcohol condition providing greater scores. There was also a main effect of time $F(1,83) = 150.59$, $p < .001$, $\eta_p^2 = .65$, with greater levels at post-drink. There was also a significant drink x time interaction $F(1,83) = 34.07$, $p < .001$, $\eta_p^2 = .58$. This was due to a nonsignificant difference between drink conditions at baseline $t(83) = .000$, $p = 1.00$, $d = 0$, but a significant difference at post-drink $t(83) = 11.70$, $p < .001$, $d = 0.91$. For irritable ratings, there was a nonsignificant main effect of drink $F(1,83) = 0.95$, $p = .332$, $\eta_p^2 = .01$, a significant main effect of time $F(1,83) = 5.203$, $p = .025$, $\eta_p^2 = .06$ with greater ratings at baseline, and a nonsignificant drink x time interaction $F(1,83) = 1.09$, $p = .299$, $\eta_p^2 = .01$. For stimulated scores, there was a nonsignificant main effect of drink $F(1,83) = .18$, $p = .676$, $\eta_p^2 < .01$. There was a significant main effect of time $F(1,83) = 6.94$, $p = .010$, $\eta_p^2 = .08$, with greater ratings post-drink. There was also a significant drink x time interaction $F(1,83) = 3.98$, $p = .049$, η_p^2 .05. This was due to stimulated ratings being higher in the placebo condition at baseline $t(83) = 1.01$, $p = .314$, $d = 0.08$ but higher ratings in the alcohol condition at post-drink $t(83) = 1.54$, $p = .129$, $d = 0.12$. For ratings of alert, there was a nonsignificant main effect of drink $F(1,83) = 3.78$, $p = .055$, $\eta_p^2 = .04$, a significant main effect of time $F(1,83) = 83.56$, $p <.001$, $\eta_p^2 = .50$, with higher ratings at baseline, and a nonsignificant drink x time interaction $F(1,83) = 1.40$, $p = .241$, $\eta_p^2 = .02$. For relaxed ratings, there was a nonsignificant main effect of drink $F(1,83) = 0.94$, $p =.336$, $\eta_p^2 = .01$, a significant main effect of time $F(1,83) = 12.79$, $p = .001$, $\eta_p^2 = .13$ with greater levels at post-drink, and a nonsignificant drink x time interaction $F(1,83) = 1.37$, $p = .246$, $\eta_p^2 =.02$. For contented ratings, there was a nonsignificant main effect of drink $F(1,83) = .26$, p

=.610, η_p^2 < .01, a nonsignificant main effect of time F(1,83) = 1.04, *p* = .310, η_p^2 = .01. There was a significant drink x time interaction F(1,83) = 6.11, *p* = .015, η_p^2 = .07. This was due to higher contented ratings in the placebo condition at baseline *t*(83) = 1.30, *p* = .196, *d* = 0.10, but higher contented ratings in the alcohol condition at post-drink *t*(83) = 1.67, *p* = .099, *d* = 0.13.